STUDY OF CHRONIC KIDNEY DISEASE IN GERITRIC POPULATION – ETIOLOGY, CLINICAL PROFILE AND OUTCOME

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CERTIFICATE

This is to certify that the dissertation titled "**STUDY OF CHRONIC KIDNEY DISEASE IN GERITRIC POPULATION – ETIOLOGY, CLINICAL PROFILE AND OUTCOME**" is the bonafide original work of in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the Tamilnadu DR. M.G.R Medical University to be held in APRIL 2016. The Period of study was from April 2015 to September 2015.

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DECLARATION

I, **Dr. GUHAN R** solemnly declare that dissertation titled "**STUDY OF CHRONIC KIDNEY DISEASE IN GERITRIC POPULATION – ETIOLOGY, CLINICAL PROFILE AND OUTCOME**" is a bonafide work done by me at Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai-3 during April 2015 to September 2015 under the guidance and supervision of my unit chief **Prof. S. TITO**, Professor of Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai.

This dissertation is submitted to Tamilnadu Dr. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – I) in General Medicine – APRIL 2015.**

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INTRODUCTION

In 2002, the National Kidney Foundation (NKF) published the Kidney Disease Outcomes Quality Initiative (KDOQI) Clinical Practice Guidelines for Chronic Kidney Disease: Evaluation, Classification, and Stratification. These clinical practice guidelines expanded the focus of chronic kidney disease (CKD) management from end-stage renal disease (ESRD) to the entire spectrum of kidney disease, from early kidney damage through to kidney failure. Guidelines, prior to this, primarily addressed hundreds of thousands patients with ESRD who were receiving dialysis. The goal of increasing the scope of diagnosis of Chronic Kidney Disease and identifying patients earlier in the course of the disease when treatment could potentially prevent the loss of kidney function and slow the progression of the disease.

These guidelines have had a substantial effect on clinical practice and as a result Chronic Kidney Disease is now identified much earlier in the disease process. Additionally, as the publication of these guidelines, several epidemiological studies have deter- mined the associations of Chronic Kidney Disease with adverse health outcomes in the general population, further highlighting the public health importance of Chronic Kidney Disease.

However, an unintended consequence of these efforts is that a very high percentage of older adults are being identified as having Chronic Kidney Disease.

The care for older adults with Chronic Kidney Disease, or geriatric nephrology, has gained recent attention and has become a focus of epidemiological research studies. With the increase in percentage of elderly population the prevalence of chronic Kidney Disease has increased and is expected to increase in the future. Further, it is important to emphasize that ageing kidney with decreasing glomerular filtration rate is different from diseased kidney with decreased glomerular filtration rate in that the diseased kidney suffers more complication. However the incidence of complications in the diseased kidney is substantially more than in the younger population. Not only are the complications specific to Chronic Kidney Disease are increased but also non specific disease conditions such as functional decline, cognitive impairment, fragility finds increased incidence in this subset of population.

In this study we analyze the spectrum of presentation of chronic kidney disease in the older population.

AIMS & OBJECTIVES

1. To analyze the spectrum of chronic kidney disease in the elderly population.

2. To analyze the etiology, duration, stage of the CKD and serum creatinine at presentation.

3. To look into the presence of smoking and alcohol consumption

4. Functional and nutritional assessment of the geriatric population including body mass index, frailty, falls, incontinence and dementia.

5. To analyze the presence of various acute and chronic complications of chronic kidney disease at presentation such as acute on chronic kidney disease, acute pulmonary edema, anemia, acidosis, hyperkalemia, hyponatremia, encephalopathy, pericarditis and correlate the occurrence of these complications with advancing stage of CKD

6. To quantify the ejection fraction, analyze ECG and chest X ray in the geriatric CKD population.

7. To analyze abnormalities in urinalysis and presence of viral markers and special investigation (if any done)

8. To analyze the mode, center, frequency, funding for renal replacement therapy

9. To observe and compare the follow up in patients from rural and urban population

REVIEW

OF

LITERATURE

“Chronic kidney disease is defined as abnormalities of kidney structure or function, present for more than 3 months, with implications for health. These include the following.

(a)Markers of kidney damage - Albuminuria (AER ≥ 30mg/24 h; ≥ 30 mg/g [≥ 3 mg/mmol]), urine sediment abnormalities, electrolyte and other abnormalities caused by tubular disorders, abnormalities detected through histology, Structural abnormalities detected through imaging, history of renal transplantation.

(b) Decreased GFR <60 ml/min/1.73m^2

as defined by National Kidney Foundation - KDIGO 2012 clinical practice guidelines for evaluation and management of chronic kidney disease”[1]

Prior to the definition for chronic kidney disease introduced and put forth by the National Kidney Foundation (NKF) Kidney Disease Outcome Qualitative Initiative (KDOQI) in 2002[2], problems pertaining to end stage renal disease were of major concern. This definition opened up new avenues and widened the perspective towards chronic kidney disease, focusing not only on the end stage renal disease, but also on the wider spectrum encompassing various stages of Chronic kidney disease. In doing so, the management in chronic kidney disease experienced a paradigm shift, now focusing on the prevention and retarding the further

progression of kidney disease, addressing complications earlier than before, preparing the patient for renal replacement therapy and renal replacement therapy.

In view of the difficulties associated with glomerular filtration rate measurement using radioactive isotopes and the difficulty associated with the 24 hour urinary collection, it is recommended that the glomerular filtration rate be estimated (eGFR) by using formulas and this eGFR has been incorporated into the staging system for chronic kidney disease. This staging system has been used in the clinical decision making and in guideline updates. Though the eGFR was most commonly estimated using the modification of diet in renal diseases (MDRD), Chronic kidney disease Epidemiology Collaboration (CKD - EPI) equation has fallen into favour after its recommendation by the KDIGO[3].

The staging of CKD as recommended by KDIGO,

GFR category	GFR (ml/min/1.73 m^2)	Terms
G1	≥90	Normal or high
G2	60-89	Mildly decreased*
G3a	45-59	Mildly to moderately decreased
G3b	30-44	Moderately to severely decreased
G4	15-29	Severely decreased
G5	<15	Kidney failure

"Table 1. GFR category in staging

*Relative to young adult level
GFR category G1 and G2 does not fulfill the criteria for CKD without any other evidence for kidney damage [1]

Category	AER (mg/24 hours)	ACR (approximate equivalent) (mg/mmol)	(mg/g)	Terms
A1	<30	<3	<30	Normal to mildly increased
A2	30-300	3-30	30-300	Moderately increased*
A3	>300	>30	>300	Severely increased**

Table 2. Albuminuria category in staging

*Relative to young adult level.
**Including nephrotic syndrome (albumin excretion usually >2200 mg/24 hours [ACR 42220 mg/g; 4220 mg/mmol])"

Prognosis of CKD by GFR and Albuminuria Categories: KDIGO 2012

GFR categories (ml/min/ 1.73 m²) Description and range				Persistent albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30-300 mg/g 3-30 mg/mmol	>300 mg/g >30 mg/mmol
	G1	Normal or high	≥90			
	G2	Mildly decreased	60-89			
	G3a	Mildly to moderately decreased	45-59			
	G3b	Moderately to severely decreased	30-44			
	G4	Severely decreased	15-29			
	G5	Kidney failure	<15			

"Table 3. Staging of CKD. Green is low risk (no CKD if there are no other markers of kidney damage); Yellow is moderately increased risk; Orange is high risk; Red is very high risk."[1]

ETIOLOGY

The various aetiologies for chronic kidney disease are diabetic nephropathy, chronic glomerulonephritis, hypertension associated chronic kidney disease, ADPKD and other cystic kidney diseases and tubulointertial diseases. The predominant cause in a population depends on the geographic area. In a patient who is newly diagnosed with chronic kidney disease and hypertension first time and no obvious causes for

glomerular and tubulointerstitial disorder could be made out, then it is probably due to hypertension

CLINCAL PRESENTATION

Patients present with symptoms only in stage 4 or 5 of CKD. Preceding which patients are usually asymptomatic and present with either symptoms and complications pertaining to systemic disease process or cardiovascular complications and other complications of CKD. Patients with chronic kidney disease may succumb to cardiovascular complications even before they progress into the advanced stages of chronic kidney disease. However there is no current recommendation for screening of general population for chronic kidney disease. Though certain guidelines recommend screening of special group of population to detect CKD in the early stages.[4]

RETARDING THE PROGRESSION OF CHRONIC KIDNEY DISEASE:

"An important step in the management of CKD is retarding the progression of disease. This includes blood pressure control, glycemic control, anti-proteinuria agents (ACE inhibitors and ARB) and dietary restrictions apart from specific treatment of the underlying disease that is

the source for kidney disease.

The degree of hypertension in CKD is associated with the rate of loss in renal function. Controlling the blood pressure retards the loss in renal function. Until recently, lower targets for blood pressure was set for the CKD population with hypertension as compared to the general hypertensive population. However in the recent turn of events it is now focussed on less intensive and individualised targets for patients with CKD[5]. JNC 8 recommends the target blood pressure in CKD patients with hypertension to be 140/90 mmHg. The pharmacotherapy should focus on the cardiovascular risk, other comorbidities and the treating physician should be considerate on the side effects of the drug in the patients being used. The blood pressure recorded at clinic can be falsely elevated in 30% patients who are normotensive and 40% patients who are tagged adequately controlled are recorded to have elevated levels of blood pressure at home. Hence when possible home based blood pressure monitoring should be done if the patient prefers.

ACE inhibitors and ARBs stand the first line therapy in patients with CKD and hypertension. They exert benefit by anti- hypertensive effect, anti- proteinuric effect and also by exerting beneficial effect on cardiovascular system. Usually multiple drugs are required for the optimal control of the blood pressure in CKD patients. However it is recommended by the KDIGO 2012 guidelines that ACE inhibitors, ARBs

and other nephrotoxic agents including potassium sparing diuretic, NSAIDs be spared from use in patients with acute on chronic kidney disease and hyperkalemia.

If adequate control of blood pressure is not achieved, a second drug may be added. If 24 hour urinary PCR is <0.6, then amlodipine may be used. If the 24 hour urinary PCR >0.6, a non dihydropyidine calcium channel blocker such as diltiazem may be used. This is based on the evidence that amlodipine causes peripheral vasodilation and increases renal blood flow increasing intra- glomerular pressure and hence increasing the proteinuria. If further adequacy of blood pressure control is required, the addition of diuretic as the third line is beneficial. The choice of diuretic depends on the patient characteristic and should be tailored to the serum creatinine, salt intake, diabetic status, arrhythmia and cardiomyopathy concerns. A detailed investigation for secondary causes of hypertension is required if 3 drug regimen does not provide blood pressure control. Beta blockers can be added along the treatment course as when they are indicated for ischemic cardiac disease and arrhythmia. However their role in treatment of hypertension has been questioned due to the wide array of metabolic disturbances it produces."[7]

Obesity and malnutrition being varied ends of a spectrum has wide implications in patients with CKD. Obesity advances the rate of progression of CKD. Hence, early in CKD when obesity may be present,

weight reduction is advisable. However as the CKD progresses, malnutrition is more common. Malnutrition occurs due to loss of appetite causing poor dietary intake, poor absorption of nutrients in a uremic gut, inflammation, oxidative stress, acidosis, and protein loss in the urine. In CKD patients restriction of proteins to 0.8 g/kg/day in patients with GFR <30 ml/min/1.73 m^2 retards the progression of CKD. However it is essential to be watchful as these patients are prone to develop malnourishment from multitude of causes as mentioned earlier which may be accentuated by the protein restriction. A major step in development of hypertension in patients with CKD is retention of salt and water. Hence restriction of sodium should be less than 5 g/day of sodium chloride. Water intake should be optimised to avoid volume overload and volume depletion. Potassium containing diet should be avoided for fear of hyperkalemia.[8]

REFERRAL TO A NEPHROLOGIST:

Referral to a nephrologist is suggested in the following:[4, 9]

1. CKD in advanced stages including the Category G4 and Category G5 CKD.
2. Persistent proteinuria with urinary albumin creatinine ratio ≥ 300 mg/g.
3. Red blood cells more than 20/ hpf which is not explainable
4. Worsening of CKD as established by either a sustained decrease in the

estimated GFR of more than 5 ml/ min/ 1.73 m^2 in a year or more than 25% drop in estimated GFR from the recorded baseline.

5. The hypertension in a CKD patient being resistant to the treatment combination of four or more drugs.
6. Inherited kidney disease
7. Recurrent nephrolithiasis
8. Extensive nephrolithiasis
9. Abnormalities in serum potassium that are persistent
10. Suspected stenosis of the renal artery

ACUTE ON CHRONIC KIDNEY DISEASE:

Whenever there is rapid rise in creatinine due to rapid loss in renal function, it can be due accelerated damage induced by underlying disease or a superimposed injury. Superimposed insult in chronic kidney disease can be due to volume reduction, drug induced insult, contrast induced injury, renal infections and systemic intercurrent illness, electrolyte disturbances such as hypercalcemia, decreased cardiac output, tachyarrythmias, obstructive uropathy, renal vein thrombosis, ischemic nephropathy. Volume reduction results due to gastrointestinal loss and excessive diuresis.

INITIATION OF RENAL REPLACEMENT THERAPY:

Generally it is necessary to plan the appropriate mode of renal replacement therapy (RRT) in patients with eGFR <20 ml/min/1.73m^2 and/or whom there is rapid progression in the loss of renal function. The mode of RRT should be discussed and planned with the patient, that is suited for his life. It has to be planned earlier before the actual requirement as mentioned above. A patient planned for hemodialysis, it takes 8-12 weeks for the AV fistula to mature. A patient planned for peritoneal dialysis, it takes time to heal and for appropriate education regarding the peritoneal dialysis. Since early renal transplantation has better outcome and results, it is essential to initiate in the waiting list before initiation of dialysis[10]. The possibility of a prospective living donor needs to be considered before initiating the patient on dialysis.

Initiating a dialysis early in the progression of CKD is not associated better surviva[11] . Hence it is essential to start renal replacement therapy as per KDIGO clinical guidelines. It is indicated when patients symptoms and signs are explainable by the failing kidney. These include

1. Acid-base and electrolyte abnormalities
2. Serositis,
3. Uncontrolled volume overload status
4. Uncontrolled hypertension
5. Worsening nutritional state despite intervention

6. Pruritus

7. Cognitive impairment

These complications positively correlate when the eGFR drops to less than 15 ml/min/1.73m^2

The common etiologies in the causation of CKD depends on population being studied. However the most common causes include diabetes mellitus, chronic glomerulonephritis. Chronic interstitial nephritis. However in developing country like India diabetes mellitus remains the most common cause of chronic kidney disease

IMPACT OF DIABETES ON KIDNEY

Diabetic nephropathy has been reported as complications in patients with Type 1 diabetes mellitus, Type 2 diabetes mellitus and other causes of diabetes also resulting from endocrine insufficiency due to a pancreatic disorder such as chronic pancreatitis or after a pancreatectomy. This implies a chronic elevation of glycemic levels in the blood for a sufficient period of time is ample and as well essential in the development of diabetic nephropathy

EPIDEMIOLOGY OF DIABETIC NEPHROPATHY

In type 1 diabetes mellitus the evolution of diabetes mellitus over the course has been well characterized. Usually a 20-30% of the patients with Type 1 Diabetes mellitus will progress to develop moderately increased proteinuria (previously termed as microalbuminuria). Under 50% of the patients with moderately increased proteinuria progress on to severely increased albuminuria (previously termed macroalbuminuria). The moderately increased albuminuria usually heralds the development of severely increased albuminuria. However severely increased albuminuria might not herald the development of advanced nephropathy with glomerular filtration rate of <60 ml/min/1.73m^2. Patients with severely increased albuminuria may have any of the following course. The proteinuria may progressively increase and progress to reduction in GFR. The severely increased albuminuria may stay the same or regress to moderately increased albuminuria or even less than moderately increased albuminuria (<30mg/day)[12-14]. Before the institution of tight glycemic control and the use of ACE inhibitors, the prevalence of albuminuria, overt nephropathy, advanced kidney disease and end stage renal disease were high. A high incidence of upto 25% to 45% of patients developing overt nephropathy with ESRD developing in upto 17% of the patients. However with the strict glycemic control and the use of ACE inhibitors,

the prevalence of overt nephropathy and ESRD has reduced[15-20]. The risk of developing severely increased proteinuria usually occurs between 10 to 15 years and in <1% of the patients between 20 to 25 years. Hence a patient developing proteinuria for the first time after 20 to 25 years of having no proteinuria, non diabetic causes of kidney disease needs to be ruled out.

Data suggest that the risk of nephropathy is equal in both Type 1 and Type 2 diabetes mellitus. This is based on Ritz et al, 1999 that the progression from onset of diabetes mellitus to proteinuria and from proteinuria to ESRD is the same in Type 1 and Type 2 diabetes mellitus [21,21]. As is the case with Type 1 DM, optimal glycemic control with use of ACE inhibitors, moderately increased albuminuria can regress retarding the progression and may even prevent the development of overt nephropathy. "The occurence of moderately increased albuminuria, severely increased albuminuria, and either increased plasma creatinine (defined as ≥2.0 mg/dL) or requirement for renal replacement therapy was 25, 5, and 0.8 percent, respectively at 10 years of follow up ashown in the UKPDS study."[23]

PATHOGENESIS:

In the pathogenesis of diabetic nephropathy, various parameters play role.

The contribution from the intraglomerular hypertension due to renovascular involvement by the disease process is evident from the use of drugs such ACE inhibitors which decrease the intraglmerular hypertension[25].

Hyperglycemia has a multitude of effects in the development of diabetic nephropathy. The increased glucose causes glycation of the mesangial matrix protein which results in mesangial expansion and apoptosis of the mesangial cell. The increased glucose in the circulation causes a non enzymatic glycation of the circulating and tissue aminoacids resulting in the formation of Advanced glycation end product. The AGEP bonds with collagen while it gets deposited in the kidney resulting in microvascular complications[27-29, 31]. Hyperglycemia also plays role by increasing the activation of protein kinase C and increased expression of heparanase increases the permeability of the filtration membrane to albumin.

Increased plasma prorenin activity, increased cytokines including VEGF, TGF beta, impaired expression of nephrin and defective podocyte specific insulin signaling also play role in the development of diabetic nephropathy[30].

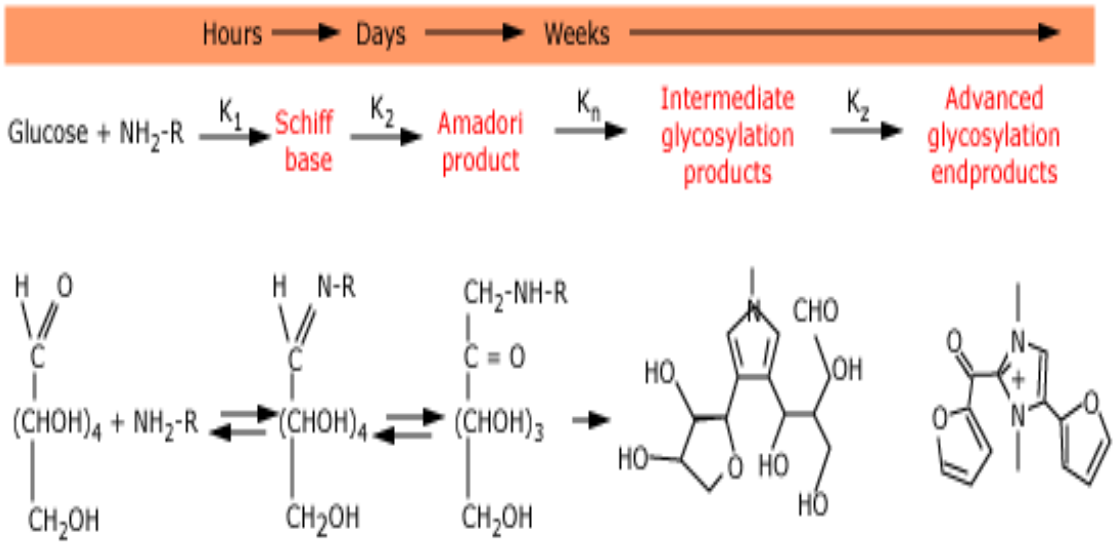

Fig 4. AGE product formation in the pathogenesis of diabetic nephropathy .

RISK FACTORS:

Various risk factors contribute to the accelerated loss of renal function in a patient with diabetic nephropathy. Genetic factors play a role in the development of diabetic nephropathy. This is evident from the fact that incidence of diabetic nephropathy is increased in those patients with family history of diabetes mellitus. “Pettitt et al assessed the likelihood of the child developing proteinuria in two successive generation in Pima Indians. He found that the incidence of developing nephropathy was 14% if none of the parents had nephropathy, 23% if one of the parent had nephropathy and 46% if both the parents had

nephropathy"[32]. Studies that have examined the effect of ACE gene genotype has produced conflicting results. However DD polymorphism in the ACE gene has been associated with the increased risk of developing diabetic nephropathy[33].

The association of age in the development of diabetic nephropathy is the increased occurrence of diabetic nephropathy as the age advances[34]. In patients with Type 1 DM, the risk of developing ESRD is cery low if diagnosed prior to the age 5[35]. Poor glycemic control, African American race, increased duration of diabetes, obesity, oral contraceptives, smoking. Increased blood pressure are associated with increased risk of development of diabetic nephropathy.

HISTOLOGY:

The histological examination of the renal tissue under light microscopy reveals measangial expansion and in advanced stages glomerular sclerosis. Examination under electron microscopy reveals glomerular basement membrane thickening. A nodular deposition of hyaline material in the arteriolar walls (Nodular Glomerulosclerosis) referred to as the kimmelstein Wilson lesion can also be detected. The prognostic implication of these lesions do not differ.

"The classification proposed by the Renal Pathology Society is as

follows:

Class I: Isolated glomerular basement membrane thickening. Basement membranes are greater than 430 nm in males older than age 9 and 395 nm in females. There is no evidence of mesangial expansion, increased mesangial matrix, or global glomerulosclerosis involving >50 percent of glomeruli.

Class II: Mild (class IIa) or severe (class IIb) mesangial expansion. A lesion is considered severe if areas of expansion larger than the mean area of a capillary lumen are present in >25 percent of the total mesangium.

Class III: At least one Kimmelstiel-Wilson lesion (nodular intercapillary glomerulosclerosis) is observed on biopsy and there is <50 percent global glomerulosclerosis.

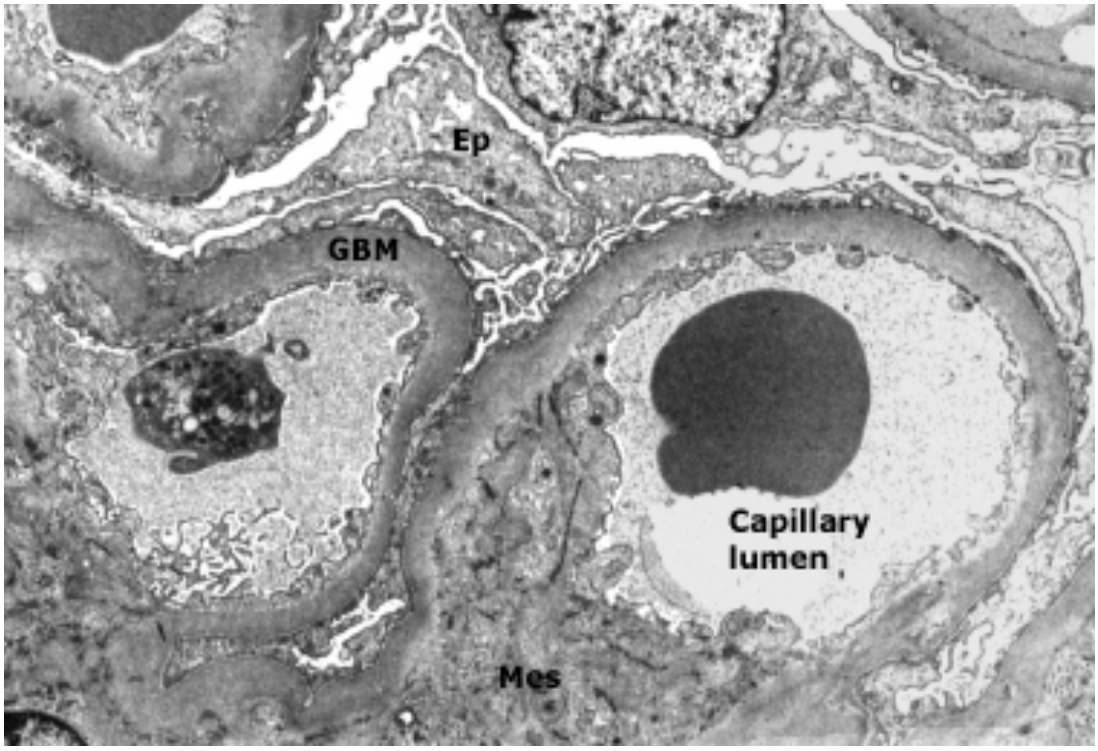

Fig 5. Electron Microscopy of glomerulus in a patient with diabetic nephropathy showing increased thickening of the basement membranes.

Class IV: Advanced diabetic sclerosis. There is >50 percent global glomerulosclerosis that attributable to diabetic nephropathy.

The severities of interstitial and vascular lesions were also assigned scores:

A score of 0 was assigned if the interstitium had no areas of interstitial fibrosis and tubular atrophy (IFTA); scores of 1, 2, or 3 were assigned if areas of IFTA <25, 25 to 50 or >50 percent, respectively.

A score of 0 was assigned if no T lymphocytes or macrophage infiltrate

was present. Scores of 1 or 2 were assigned if infiltrate was limited to the area surrounding atrophic tubules, or if infiltrate was not limited, respectively.

Scores of 0, 1, or 2 were assigned if no arteriolar hyalinosis, one arteriole, or more than one arteriole with hyalinosis was present. In addition, the most severely affected arteriole was assigned a score of 0, 1, or 2 if there was no intimal thickening, intimal thickening < thickness of media or intimal thickening > thickness of the media."[3]

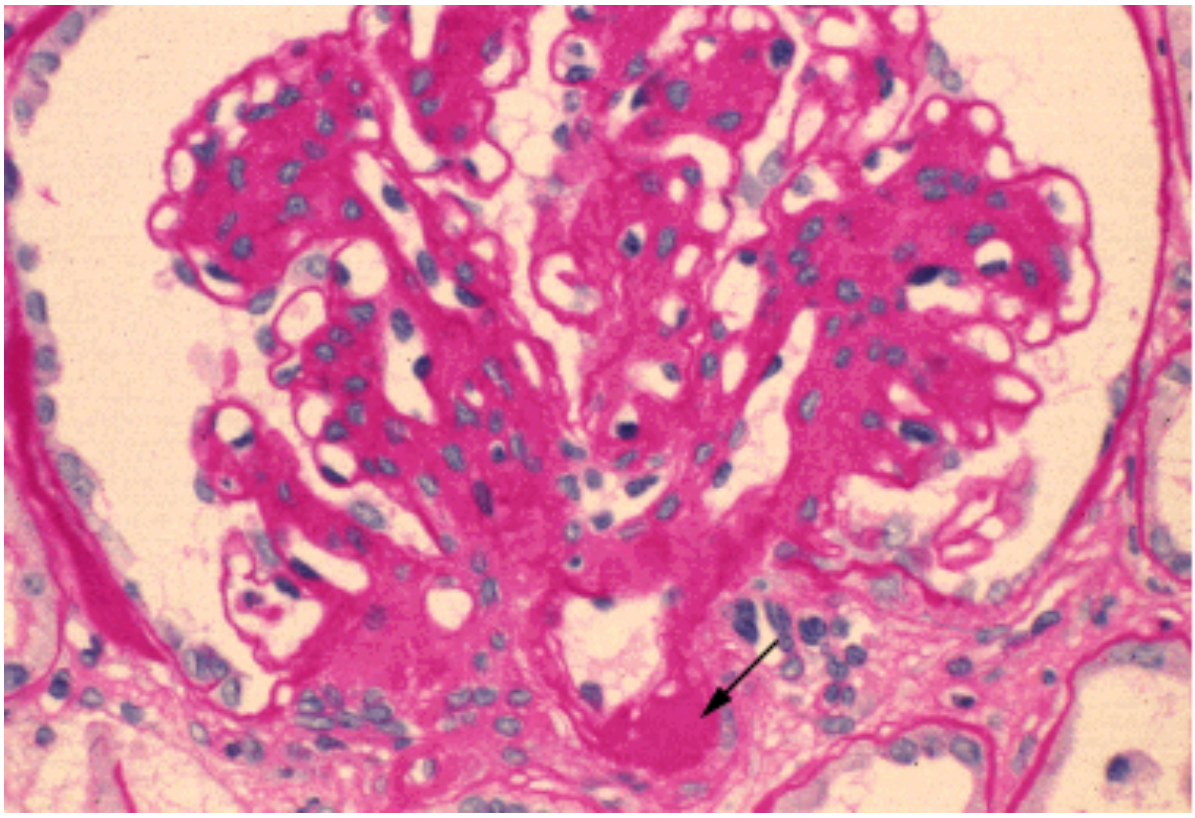

Fig 6. Light microscopy of glomerulus showing hyaline thickening at the walls of the arteriole (arrow) with diffuse mesangial expansion.

Other causes of nodular glomerulosclerosis are

1. Amyloidosis
2. Monoclonal immunoglobulin deposit disease, with majority due to kappa light chain deposit disease and other organized glomerular deposition disease
3. Fibrillary and immunotactoid glomerulonephritis
4. Fibronectin glomerulopathy
5. Collagen III glomerulopathy
6. Chronic hypoxic conditions including cyanotic congenital heart disease
7. Chronic ischemic conditions such as renal artery stenosis
8. Chronic membranoproliferative glomerulonephritis Type I
9. Idiopathic glomerulosclerosis the occurrence of which can be seen in smokers and hypertensive patients. It can also be seen in patients with metabolic syndrome without overt diabetes mellitus.

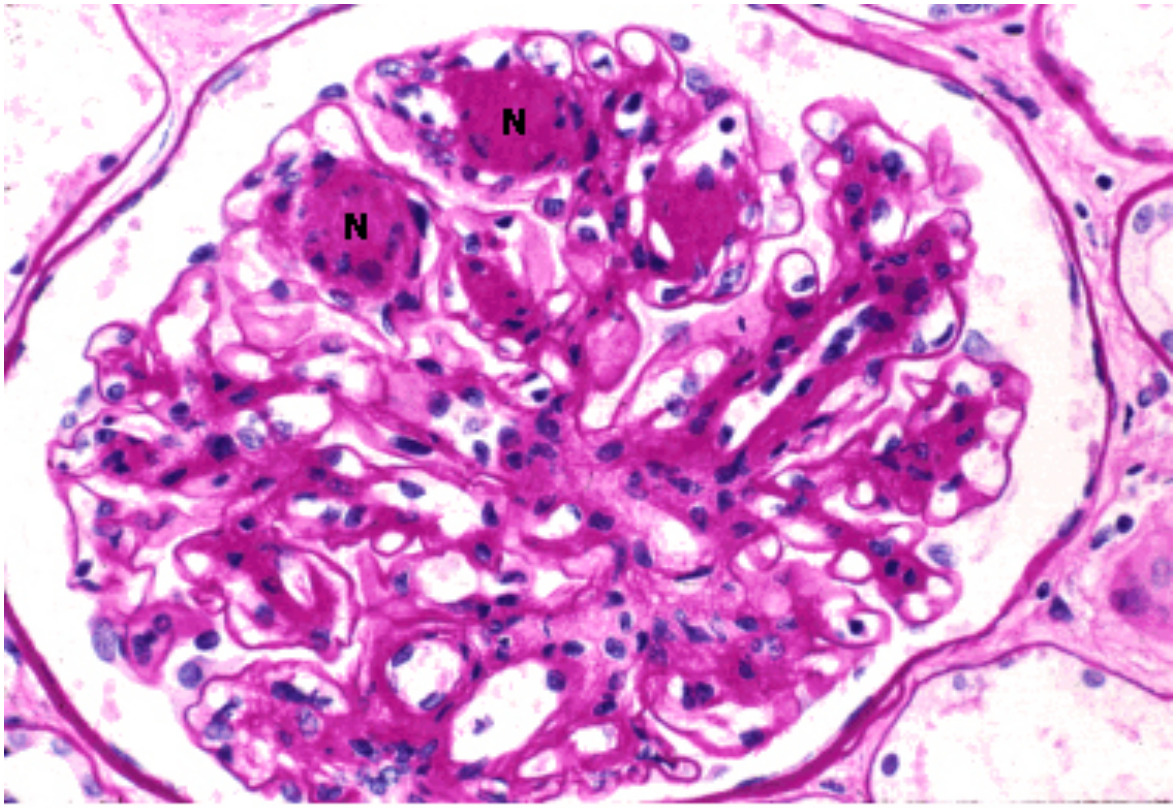

Fig 7. Light microscopy showing nodular (N) glomerulosclerosis in patients with diabetic nephropathy

CLINICAL PRESENTATION AND LABORATORY FINDINGS:

The clinical presentation in diabetic nephropathy includes albuminuria and hematuria. The albuminuria in diabetic nephropathy can be rather moderately increased albuminuria or severely increased albuminuria. Moderately increased albuminuria heralds the occurrence of

severely increased albuminuria. However the degree of albuminuria in severely increased albuminuria poorly correlates with the progression of renal injury[37]. Despite a decreasing albuminuria the patient may progress to advanced kidney disease and may progress on to ESRD. But patients with the highest rate of severely increased albuminuria had the increased loss renal function[38]. The reason for progressive loss of renal function in diabetic patients without proteinuria is not known but it maybe attributed to the renovascular disease in such patients. However the resistance offered to blood flow in both proteinuric diabetic nephropathy and non proteinuric diabetic nephropathy as detected by renal duplex scan remains the same. The occurrence of such non proteinuric diabetic nephropathy is increased in the younger population in whom the prevalence of such renovascular involvement is less common. These above mentioned points argues against the renovascular involvement as a cause of renal disease progression in non proteinuric diabetic nephropathy

The presence of microscopic hematuria has been detected in diabetic nephropathy though the sediments in the urine may not be detected. However, hematuria in a diabetic individual can also be due to other glomerular disease such as membranous nephropathy, IgA nephropathy. The presence of microscopic hematuria in a series of evaluated patients was due to IgA nephropathy, membranous nephropathy and severe diabetic nephropathy[39]. Similarly, evaluation of a patient series with red

cell cast revealed IgA nephropathy in 2 patients, post infectious glomerulonephritis in 1 patient and diabetic nephropathy a the cause in other 5 patients[40].

DIABETIC NEPHROPATHY & DIABETIC RETINOPATHY – THE LINK:

Patients with Type 1 DM and diabetic nephropathy will have other associated microvascular complications such as retinopathy. The occurrence of retinopathy will precede the development of nephropathy. Combining the facts, a diabetic patient with renal involvement but without retinopathy, non diabetic causes of the renal involvement should be considered. However, the involvement of retina by the diabetes may occur isolated without the development of nephropathy

The scenario of the link between the retinopathy and nephropathy in type 2 DM is not the same. Only about 56% of the diabetic nephropathy patients were complicated by retinopathy however none of the patients with non diabetic etiology as a cause for nephropathy had retinopathy[41]. Hence the presence of retinopathy has a 100% positive predictive value and a low negative predictive value. Treatment of nephropathy with the blockade of the angiotensin system has similar beneficial effects on the retinopathy[42].

NON DIABETIC INVOLVEMENT OF KIDNEY IN DIABETIC INDIVIDUALS:

It is essential to consider the presence of a non diabetic etiology in a diabetic individual in the following scenarios;

1. The presence of renal involvement without the presence of retinopathy in Type 1 DM and the occurrence of proteinuria usually within 5 years from the onset of diabetes in Type 1 DM. However, in Type 2 DM it is difficult to establish with certainty the onset of the diabetes and henceforth the time of proteinuria since the onset[43].
2. The presence of a sediment cast in the urine microscopic examination. Though red cell cast can be present in severe diabetic nephropathy, its presence warrants the evaluation of non diabetic causes.[45]
3. The presence of other features in systemic examination that points towards secondary causes[44].
4. The rapidity with which the renal function worsens. The diabetic involvement of the kidney usually progresses insidiously. Hence an acute or rapid decline in renal function in a diabetic patient; causes other than diabetic nephropathy should be ruled out.
5. The rapid decline in GFR of more than 30% from the baseline within a stipulated time period of 2 to 3 months following the administration of

ACE inhibitors also requires renovascular causes of declining renal function to be evaluated[44]. In such patients nephrosclerosis secondary to diabetes especially in older individuals must also be considered

Hence the above mentioned patients should be evaluated with renal biopsy and other clinically warranted investigations.

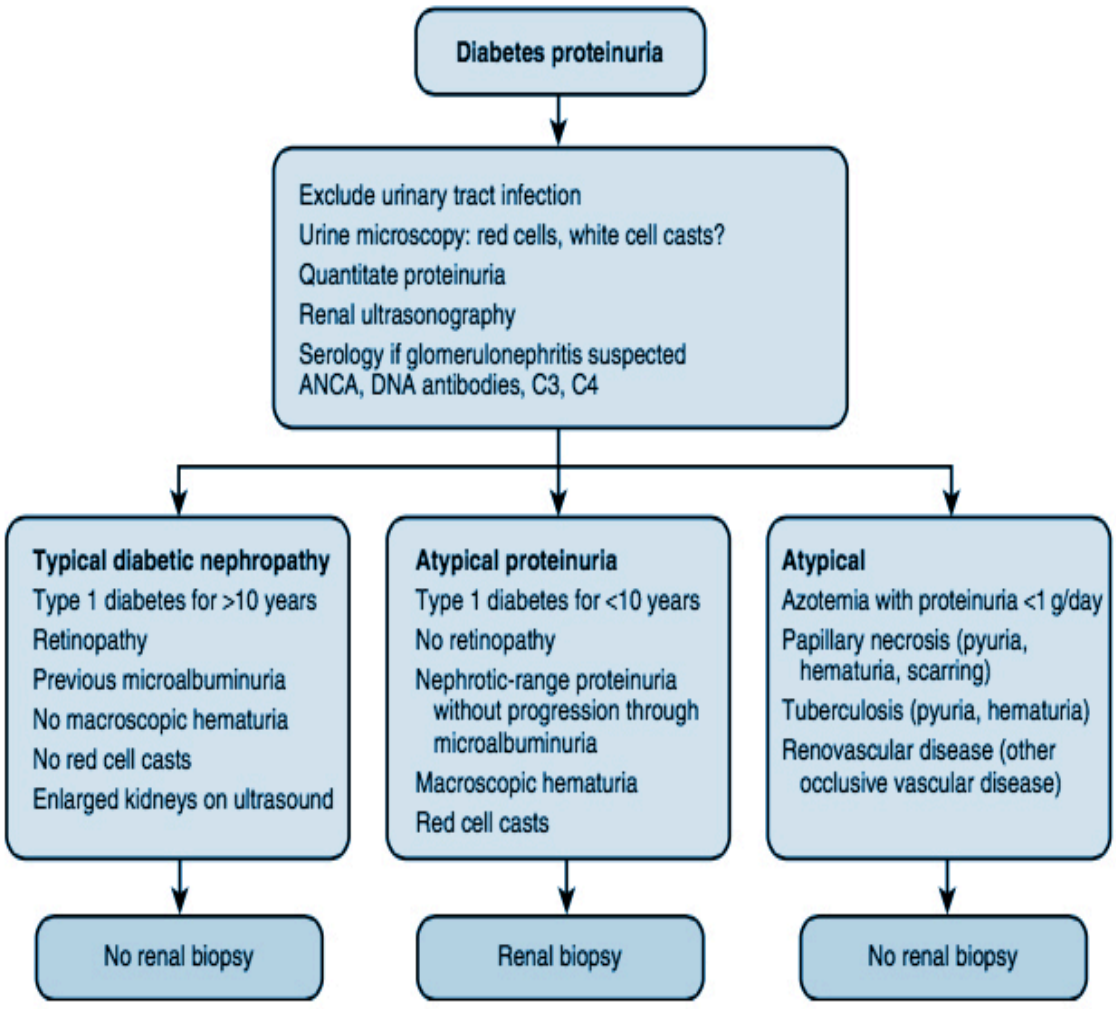

Fig 8. Flow diagram depicting evaluation of diabetic nephropathy

TREATMENT OF DIABETIC NEPHROPATHY:

The treatment of diabetic nephropathy involves a multidirectional approach

GLYCEMIC CONTROL:

Achieving a good glycemic control prevents or retards the onset of development of diabetic nephropathy and reverses the biochemical and pathological changes in diabetic nephropathy. Pathological changes such as mesangial matrix expansion had experienced volume regression. Biochemical changes including proteinuria had decreased and even returned to normal[46]. However, the biochemical improvement occurred 2 years following a strict glycemic control[47, 48] and the pathological improvement occurred 10 years following pancreatic transplantation in Type 1 DM[49, 50]

DRUGS:

ANGIOTENSIN INHIBTIORS:

The angiotensin inhibitors have produced dramatic effects in the treatment of diabetic nephropathy. The benefits demonstrated by the angiotensin inhibitors is beyond that could be explained by just reduction in blood pressure. The additional mechanism by which it improves renal

function is by its anti-proteinuric effect. The anti-proteinuric effect is produced by reducing the intraglomerular hypertension. The benefit derived from ACE inhibitors could also be explained by additional mechanisms which include interfering in the actions of transforming growth factoe β. The use of ACE inhibitors has the maximum benefit when used in the stage of moderately increased albuminuria in which it can revert the albuminuria and also retard the progression to overt nephropathy[51, 52]. When used in patients with severely increased albuminuria, it decreases the proteinuria nad retards the progression of renal loss[53, 54].

Though the benefit of ACE inhibitors has been markedly reduced in patients with advance kidney disease and ESRD, the rate of rise in creatinine may be reduced.

The benefit of ACE inhibitors is maximally studied in diabetic nephropathy due to Type 1 DM. Data on its efficacy in diabetic nephropathy due to Type 2 DM is limited. Although benefit similar to Type 1 DM is expected to be present

The beneficial effect on the use of Angiotensin receptor blockers and its renoprotective effect has been demonstrated in two trials[55, 56]. Decreasing the albuminuria using these drugs reduced the adverse cardiovascular events when such drugs were used in the first 6 months of diabetic nephropathy. Combination of ACE inhibitors and angiotensin

receptor blockers produced reduction in proteinuria that could be explained by the use of single drug alone as monotherapy[57, 58]. However, use of such combination is associated with increased adverse effects and increased hospitalization due to increased rate of acute kidney injury. Hence until further trials clearly demonstrating beneficial effects that outweigh the side effects are available, this combination is not recommended[59].

ALISKIREN AND ANGIOTENSIN INHIBITORS:

The combination of renin inhibitors with angiotensin inhibitors does not have anybeneficial effects. Rather it is associated with increased risk of adverse events as demonstrated in the ALTITUDE trial[60].

BARDOXOLONE METHYL:

Bardoxolone methyl is an antioxidant inflammatory molecule. Though the benefits of the drug has been explained in animal models of acute kidney injury, it is not recommended for use in diabetic nephropathy as suggested by data derived from BEAM trial[61].

PENTOXIFYLLINE:

Pentoxifylline is a non specific phosphodiesterase inhibitor and it has been used with success in the treatment of peripheral vascular disease

and in severe alcoholic hepatitis when the maddreys discriminant function scoring is more than 32. The additional benefit of using pentoxifylline in the treatment of alcoholic hepatitis over steroids was the renoprotective effect attributed to the pentoxifylline decreasing the incidence of renal failure. Similarly the effects of pentoxifylline was extrapolated for use in diabetic nephropathy and in a study, it was demonstrated that the drug either improves or stabilizes the GFR. However further studies are required to prove its efficacy before recommendation in the treatment of diabetic nephropathy[62].

CALCIUM CHANNEL BLOCKERS:

The use of non dihydropyridine calcium channel blockers in experimental models has shown that it decreases proteinuria. However, it was associated with increased degree of interstitial fibrosis and global sclerosis. The adverse effects observed with the non dihydropyrindine calcium channel blockers can be ameliorated by its combination with angiotensin inhibitors[63]. A similar effect on proteinuria was not associated with the use of dihydropyridine calcium channel blockers such as amlodipine. Rather, these drugs increase the rate of protein excretion.

ALDOSTERONE ANTAGONIST:

The use of aldosterone antagonist in patients on ACE inhibitors has an additive effect on decreasing proteinuria[64,65]. However, the use of this combination in clinical practice is associated with increased risk of hyperkalemia. Hence it is necessary to advice a strict and stringent potassium restriction in addition to salt and potassium restriction.

PROTEIN RESTRICTION:

The reduction in intake of protein (and phosphorus) in diet to 0.6g/kg/day markedly retards the rate at which GFR drops. In a study it was noted that protein restriction to 0.6 g/kg/day was found to have a 75% improvement in the GFR decline[66, 67]. Further benefits of restricted diet such as decreased mortality and decreased onset of ESRD were noted in the low protein diet arm[68]. However, the exact mechanism remains unclear.

Protein restriction in these patients are also associated with increased of malnutrition due to associated restriction in the intake of carbohydrate and fat and due to augmented protein catabolism in diabetics attributed to insulin deficiency[69].

It is well known data that proteinuria is a major factor that is responsible for progressive renal injury and advancement of CKD. This is attributed to the increasing interstitial fibrosis that is associated with

increasing degrees of proteinuria. A patient who has a 50% reduction in proteinuria in response to angiotensin inhibitors usually have a favorable prognosis. This was evident from IDNT and RENAAL trials. Increasing the reduction in proteinuria is associated with proportionate decrease in the loss of renal function[70].

SALT RESTRICTION:

Increasing intake of salt has been associated with decreased response of proteinuria to ACE inhibitors. Restriction of salt augments the response to ACE inhibitors by further decreasing proteinuria in patients with non diabetic kidney disease. Similar effect of augmented response to ACE inhibitors was also observed in diabetic nephropathy[71, 72]. Patients who fail to respond to respond to ACE inhibitors despite a drop in blood pressure may be poorly compliant with salt intake.

A daily salt intake of <70 meq/ day is associated with morbidity and mortality benefit. However this degree of salt restriction is not practically feasible. Hence a salt restriction of < 100 meq/ day is advisable.

OTHER THERAPIES:

A reduction in weight by physical activity in patients with obesity and lowering the cholesterol levels in patients with hypercholesterolemia is associated with additional benefit in diabetic nephropathy.

A multidirectional approach is hence required to treat patients with diabetic nephropathy.

COMPLICATIONS OF CKD:

The complications of CKD include both acute and chronic:

1. Anemia and other hematological abnormalities
2. Acid- base disturbances including acidosis and hyperkalemia
3. Volume overloaded state
4. Renal Osteodystrophy
5. Dyslipidemias
6. Malnutrition
7. Acute on Chronic Kidney disease
8. Accelerated cardiovascular disease
9. Central and peripheral nervous system complications

Cardiovascular disease remains the most common cause of death in CKD patients. The incidence of cardiovascular complication in a CKD patient is 10 times more common than the general population[73]. The patient may reach advanced stages of cardiovascular disease even before the end stage renal disease is reached. The patient may not even reach the

ESRD stage due to cardiovascular death. The enhanced cardiovascular mortality is related to the increase in ischemic vascular disease and hypertension. The ischemic vascular disease is increased due to traditional risk factors such as diabetes mellitus, hypertension and hypercholesterolemia and also due to CKD related factors such as hyperparathyroidism, hyperphosphatemia and the inflammatory milieu attributed to CKD. These factors accelerate the atherosclerotic process leading on to increased incidences of heart failure and myocardial infarction[74]. The diagnosis of myocardial infarction is further complicated by the fact that troponin T levels may be elevated in CKD in the absence of myocardial ischemia. Hence it is required to document a rise in the troponin levels in patients suspected to have myocardial infarction. However, patients with elevated levels of troponin T levels without myocardial infarction are at increased risk of cardiovascular complications. The presence of hypertension in CKD is one of the most common complication. The salt and water retention, enhanced activity of the renin angiotensin axis are the major mechanisms for the development of hypertension which can be further augmented by the use of erythropoietic agents in CKD. In addition, renovascular disease may also contribute to the development of hypertension. Hypertension has implication in both cardiovascular system and in CKD. In cardiovascular system it causes left ventricular hypertrophy and heart failure. The left

ventricular dysfunction is both systolic and diastolic dysfunction. However, the presence of normal blood pressure or decreased blood pressure in advanced stages of CKD suggests poor left ventricular function. Ischemic factors combined with hypertensive remodeling in volume over loaded patient leads to failing heart and pulmonary edema. Failure can be augmented by the presence of anemia and AV fistula leading to high output heart failure. Sometimes, low pressure pulmonary edema can be due to uremia induced leaky pulmonary vasculature which is evidence by its improvement with hemodialysis.

The treatment of hypertension is very essential in retarding the progression of CKD and heart failure. However the target blood pressure remains controversial. The current consensus for target blood pressure includes 130/80 mmHg in CKD with proteinuria and 140/90 mmHg in CKD without proteinuria. ACE inhibitors and angiotensin receptor blockers play a major role and added benefit in also reducing the intraglomerular pressure and retarding the progression of CKD. Salt restriction, fluid restriction and use of a kaliuretic diuretic may be beneficial and usually multiple drugs will be required to achieve target blood pressure in patients with hypertension in CKD. However the use of ACE inhibitors should be cautious because of increased risk of hyperkalemia and acute on CKD may be precipitated in patients with renovascular disease. Potassium sparing diuretics should be used with

caution or avoided altogether in most patients. Lipid lowering medications in the presence of hypercholesterolemia though the data on use of statins in CKD is limited and benefits of statins in advanced CKD is less clear. Regular exercise and other addressing other modifiable risk factors are essential. The presence of pericariditis either symptomatic or asymptomatic is an indication for either intensification or commencement of renal replacement therapy. Heparin should not be used in hemodialysis for uremic pericarditis as it increases the chances of bleed into the pericardial fluid. Drainage of pericardial fluid should be considered in patients with recurrent pericardial effusion.

Anemia in CKD patients starts to develop in the third stage of CKD and becomes almost universal by the fourth stage. It is usually a normocytic normochromic anemia due to decreased production due to erythropoietin deficiency. Other factors contributing to the development of anemia in CKD includes deficiency of nutritional factors such as iron, folate and vitamin B12, bone marrow fibrosis due to hyperparathyroidism, bleeding diathesis, decreased red blood corpuscle survival, chronic inflammatory state, hemoglobinopathy and the presence of comorbid factors[76]. Clinical manifestations due to anemia include cardiovascular symptoms and when severe enough results in heart failure, poor cognitive function, impaired defense against host function, growth restriction in children. Poor response of anemia to erythropoietic

stimulating agents denotes poor prognosis. These patients are at increased risk of bleeding tendency due to decreased platelet aggregation, decreased consumption of prothrombin and impaired activity of platelet factor III. The CKD patients have greater risk for thromboembolism especially in patients with nephrotic range proteinuria due to loss of anticoagulants in urine.

The use of erythropoietic stimulating agents has revolutionized the treatment of anemia in CKD reducing the requirements of blood transfusion thereby reducing the complications associated with blood transfusion including transfusion associated infections, iron overload and increased increased sensitization to donor antigens increasing the risk of graft failure[77]. After ensuring adequate iron stores evidenced by serum ferritin, vitamin B12 and folate patients are treated with erythropoietic stimulating agents. If the patient is deficient in iron, IV iron is given to the patient to replace the iron stores. It is essential to note that these patients are at increased risk of bacterial infections. Anemia resistant to treatment with recommended doses of erythropoietic stimulating agents indicate either acute and chronic inflammation, inadequate dialysis, blood loss or hemolysis, severe hyperparathyroidism and presence of chronic infections. The use of erythropoietic stimulating agents have improved the hemoglobin but there was no improvement in the cardiovascular outcomes[78]. The use of these agents are associated with increased

thrombo embolic events and increased rate at which the patients progress to dialysis[75]. More studies are required for solving these issues. The target hemoglobin in CKD is 10 - 11.5 g/dl. The uremic platelet dysfunction is treated with desmopressin and optimal dialysis[79]. It is also worth noting that the favorable risk benefit profile applicable in the general population do not apply to the CKD population due to altered balance between the procoagulant and anticoagulant factors. And when required it is prudent to use unfractionated heparin than to use low molecular weight heparin. The newer oral anticoagulants have to be assessed for dosage as per the eGFR.

The **fluid and electrolyte disturbances** in CKD alters the internal milieu resulting in various complications. There is retention of salt and water resulting in volume overloaded state. This is especially the case when the dietary sodium exceeds the ability of the kidney to clear sodium. And as long as the kidney eliminates free water that the patient consumes there is no hyponatremia. Hence hyponatremia is less common in CKD and responds to fluid restriction. The volume overloaded state can be treated using diuretics including metalazone and loop diuretics. The state of resistance to loop diuretics in CKD suggests that higher doses of loop diuretics may be required. Fluid restriction is advisable only when there is hyponatremia. Volume overloaded state not responding to diuretics is an indication for renal replacement therapy.

Further in these patients there is impairment in the ability of the tubules to reabsorb sodium and hence in volume depleted states such as GI loss and overzealous use of diuretics the contracted plasma volume can impair renal function. The proper fluid therapy in such cases can restore the renal function.

The hyperkalemia in CKD occurs with advanced stages of CKD especially when associated with high potassium diet, acidosis, catabolic state, use of potassium sparing diuretics, angiotensin inhibitors, hemolysis, blood transfusion. However, in patients with diabetes, sickle cell disease, obstructive uropathy hyperkalemia can occur at an earlier stage due to hyporeninemic hypoaldosteronism. Hyperkalemia is managed by dietary restriction of potassium and use of diuretics that favors potassium excretion and sodium polystyrene that sequesters GI potassium and favors GI loss.

The acidosis in initial stages of CKD is characterized by normal anion gap. It is due to impaired hydrogen ion excretion, not due to impaired acidification, but due to impaired ammonia production which is further depressed by the presence of hyperkalemia. The advanced stages of CKD is characterized by high anion gap. The bicarbonate level less than 20 – 23 mmol/L is associated with increased rate of catabolism in the body. This is prevented by sodium bicarbonate supplementation which retards the progression of CKD.

The **disorders of calcium and phosphate metabolism** affects the bone vascular bed and soft tissues. In advanced CKD as the eGFR drops to <60 ml/min/1.73m^2, the phosphate level in the blood increases leading to increased levels of FGF – 23. It results in decreased calcitriol and hence decreased serum calcium levels which results in elevated parathormone levels. The spectrum of bone disorder ranges from high turnover to low turn over bone disease[80]. The result of elevated parathormone in an untreated patient results in high turn over from the bone resulting in osteitis fibroa cystica resulting in the formation of bone cyst which may become hemorrhagic leading to the name brown tumor. It may also lead to bone marrow fibrosis. However, treating the patient with calcium based phosphate binders such as calcium carbonate and calcium acetate results in hypercalcemia and decreased parathormone levels and hence the increased calcium gets deposited in the vascular bed and soft tissues[81]. Decreased mineralization of the bone results in adynamic bone disease resulting in fractures[82]. Hence the use of non calcium based phosphate binders including sevalamer and lanthanum avoids the complication of adynamic bone disease and soft tissue calcification, The use of calcitriol may be given to increase the calcium levels which results in suppression of parathormone. These patients needs to be monitored for hyperphophatemia. Hyperphosphatemia can be manages using phosphate restricted diet and phosphate binders. FGF 23 levels in these patients also

increase cardiomuscular morbidity because it can cause cardiac fibrosis. Calciphylaxis is a dreaded complication in advanced stages of CKD. It results in ischemic necrosis of the skin which is preceded by livedo reticularis. It occurs in patients with severe hyperparathyroidism, use of oral calcium based phosphate binders and use of warfarin. Hence when it occurs warfarin if used should be switched to other drugs.

Other complications in CKD include peripheral neuropathy which is initially sensory and subsequently progresses to motor involvement can occur. Other neuromuscular abnormalities may also occur[83]. The use of renal replacement therapy may reverse the neuropathy especially when done before motor component occurs. Gastrointestinal abnormalities due to CKD results in uremic gastritis which may result in vomiting, abdominal pain[84]. The patient may also have dysgeusia. Malnutrition is common in CKD attributed due to poor absorption by the uremic gut and due to increased protein catabolism in these patients. In women, estrogen level decreases, causes menstrual abnormalities, infertility and increased rates of spontaneous abortion. Only 20% of patients with advanced CKD give rise to live births. In males it results in sexual dysfunction and oligospermia. Dermatologic abnormalities include pruritus in uremic patients and when gadolinium contrast is administered in CKD patients it may result in nephrogenic fibrosing dermopathy which is highly fatal[85].

CKD IN THE ELDERLY POPULATION

The CKD in the elderly has wide spread implications. Firstly, the geriatric population in the developed and the developing middle and low income countries has increased due to decreased deaths owing to the advances in the field of therapeutics. As a result, old age population contribute to a significant proportion of CKD in the general population. This is evident from the increase in prevalence of CKD from 37% to 47% in the aged population >70 years' age[86].

With the introduction of definition by the National Kidney Foundation – Kidney Disease Outcome: Qualitative initiative in 2002, there was a paradigm shift in the perspective and approach to CKD. Individuals at risk of ESRD were identified much earlier and preventive measures were taken to retard the progression of CKD. However, in the process large proportion of old age individuals were detected as CKD. This created two different perspectives. First perspective was, this definition has brought out the epidemic of CKD which was under reported. The individuals who were identified by the process were at increased risk of adverse cardiovascular events and physical functioning which could be addressed to improve morbidity and mortality. The second perspective is, the definition without intending to has over

diagnosed CKD. This resulted from failure to differentiate between chronic kidney disease and the senescence of normal kidneys. Also the use of formulas in the elderly population has been less validated and hence more patients without CKD may be diagnosed with the disease.

ESTIMATION OF GFR – FALLACIES IN OLD AGE:

The estimation of GFR came into vogue due to the cumbersome procedure of collecting 24 hour urinary sample for calculation of creatinine clearance to calculate GFR. Initially estimation of GFR was done using Cockroft Gault formula. Subsequently the formula introduced by the Modification of Diet in Renal Diseases (MDRD) study provided better results in estimating eGFR than cockroft gault formula. However the eGFR estimated by the MDRD formula correlated better in the lower range of GFR but not so in the higher ranges. Hence the CKD – EPI formula was subsequently used which has better correlation with GFR than the MDRD formula. The advantage of CKD-EPI formula was that it did not over estimate the number of patients having CKD. However it did not hold good in the elderly population in whom it over estimated the prevalence of CKD[87].

Hence the shift in marker from the use of creatinine in estimating GFR to using cystatin C. The formula containing estimation of GFR using both cystatin C and creatinine is considered best. The use of

cystatin C has the following advantages. Cystatin C sources from nucleated cells. It is filtered across the glomerulus reabsorbed in the proximal tubules and is broken down. Cystatin C is not dependent on muscle mass. It is less affected by gender, race and age. It correlates better and linearly with cardiovascular adverse outcomes than creatinine. Hence forth, estimation of GFR using formulas derived from cystatin C and creatinine has the best prediction. It is limited by its availability though it is expected to increase in availability[88].

Hence it is recommended by the KDIGO that when a patient is diagnosed as CKD due to GFR < 60 ml/min/ 1.73 m^2 with no other markers of renal damage and has been classified as Stage 3A according to the GFR, it is essential that the GFR be estimated by using formulas using creatinine and cystatin C. If the calculated GFR is >60 ml/min/1.73 m^2, then the patient does not have CKD. In this manner it was found that a large number of older individuals were reclassified from the Stage 3A to having no CKD.

THE PROGRESSION OF CKD VERSUS RENAL SENESCENCE:

The glomerular filtration rate rises to maximum during life at 4th decade and subsequently it starts to drop after at a rate of 8 ml/min/1.73m^2. It is to be considered that the decline in GFR is an ageing process and should not be confused with CKD. Baltimore longitudinal

study included patients for monitoring the creatinine clearance for estimating GFR[89]. The included patients did not have any comorbidities, renal disease or drug intake that could interfere with GFR and its estimation. It was found that the GFR progressively decreased with age. However it was the average GFR of the study population that decreased, though a subset of patient population did not have a decline on GFR suggesting an inter individual variation in GFR decline[89, 90].

RISK FACTORS IN CKD PROGRESSION:

Compared to the younger individuals, the risk factors of diabetes mellitus, systemic hypertension plays a major role in etiology and progression of CKD. Sufficient time would have lapsed following the onset of diabetes mellitus, systemic hypertension which are the main determinants in the progression of CKD. Other risk factors such as renovascular disease, obesity, hypercholesterolemia may also play a role in this population. Smoking, family history and cardiovascular diseases also plays role.

HYPERTENSION:

Systolic component of blood pressure is a major determinant in the progressin of CKD and correlated with it linearly. However the target blood pressure in the treatment of CKD patients has been controversial.

The recommendation by the Joint National Committee 8 in individuals aged more than 60 years of age is 150 / 90 mmHg of blood pressure. However, it has been documented that in patients with proteinuria, a lower target for systolic blood pressure retards the progression of blood pressure. Hence KDIGO recommends that blood pressure target in patients without proteinuria is 140/90 mmHg. The blood pressure target in patients with proteinuria (albuminuria >30 mg/dl) is 130/80 mmHg.However it is also to be noted that older individuals are more prone for side effects such as postural hyotensionwith the use of antihypertensives.

ACE inhibitors / ARBS are preferred as antihypertensive in individuals with proteinuria due to anti-proteinuric effect. However the safety data regarding the side effect profile of ACE inhibitors and ARBS are limited in the elderly population and is controversial. In RENAAL study it has been shown there was no increased incidence of hyperkalemia in older patients and it prevented the progression to ESRD in 50% patients[91]. However, in a study, use of Lisinopril in an older indivuals with an eGFR of <60 ml/min/1.73m^2 was associated with an increased incidence of hyperkalemia[92]. In AASK trial, the occurrence of hyperkalemia was more common in the ageing population. Hence, KDIGO recommends that ACE inhibitors / ARBs be used in diabetic

patients with albuminuria >30 g/day and in non diabetic patients with albuminuris >300 g/ day

DIABETES:

Diabetes is a major contributor to CKD in the elderly. The correlation between glycemic control and renal complications were evaluated and it was found that tight glycemic control was decrease in the degree of albuminuria. However, it was not clear if it retarded the progression in CKD. And it was further associated with increased incidence of hypoglycemia, weight gain, arrhythmias and increased morbidity in the tight control arm. The risk of hypoglycemia was more in the diabetic CKD which was more in diabetic patients without CKD which was in turn more than non diabetic CKD. Hence KDIGO recommends a target HbA1C of <7% and it should be higher than 7% for fear of complications in individuals with HbA1C >7%.

ACUTE KIDNEY INJURY:

AKI is considered as risk for development of CKD and its progression, especially in older individuals because of poor renal reserve in this population. Hence a sudden drop in functioning glomerular tissue would increase the delivery of blood to other functioning glomerulus which would increase the intraglomerular hypertension further worsening

the functional status of the kidney. Further, CKD patients are at more risk of developing acute worsening of renal function. NSAIDs is a common cause and hence should be avoided in patients with CKD. Also many drugs having renal excretion and older patients on multiple drugs, it is essential to consider drug to drug interaction and appropriate dosing of drugs in this patient population and hence at increased risk of medicine related complications.

NON RENAL COMPLICATIONS IN AGEING KIDNEY:

CARDIOVASCULAR OUTCOME:

CKD patients are at three times higher risk of developing adverse cardiovascular events and cerebrovascular accidents. This is especially prevalent in the patients with CKD stage 3 or higher. As suggested by the cardiovascular health study index, participants with CKD and microalbuminria had 2.5 times higher risk of myocardial infarction[93].

Patients with age >65 years, even mildly increased serum creatinine was associated with increased cardiovascular mortality as compared with individuals with normal serum creatinine[94]. Statins use decrease adverse cardiovascular events in CKD patients. However, this

effect has been documented only on patients who are not on hemodialysis and not inpatients who are on hemodialysis.

COGNITIVE IMPAIRMENT & PHYSICAL FUNCTIONING:

Cognitive dysfunction is increased in elderly patients with CKD. ESRD and increasing age are independent risk factors for cognitive dysfunction. The areas affected in cognitive testing include attention and executive function[95]. CKD in elderly increases the risk of frailty. Frailty increases disability. Disability increases admissions to nursing old age homes. CKD patients can have low levels of physical activity.

BONE DISEASE:

The metabolic bone disease results due to milieu changes secondary to CKD. Early CKD is associated with increase in FGF 23 and decreased 1,25 dihydroxy vitamin D3. As CKD progresses parathormone and phosphorus levels increase in advance stages of CKD. The predominant skeletal finding in CKD patients are osteoporosis. However it has not been found that CKD decreased bone mineral density. However it predicts that the bone loss is increased, If the bone mineral density is low, the rate of fracture is increased in both dialyzing and non dialyzing CKD. The drugs for osteoporosis can be prescribed if eGFR <30 ml/min/1.73m^2.

PROGNOSIS IN OLD AGE:

Although the prevalence of CKD is increased in older age, it is not associated with increased progression to ESRD. This is attributed to early death due to other complications of CKD in the elderly population and due to over diagnosis of CKD. For any given GFR the elderly patients are at more risk of dying than the young individuals[96].

REFERRAL TO A NEPHROLOGIST:

It is necessary to consider referral to a nephrologist in the following scenarios:

1. Nephrotic syndrome
2. Proteinuria which is less than 1 g in a non diabetic kidney disease
3. Non urologic hematuria
4. Recurrent hyperkalemia
5. Rapid decline in eGFR >5ml/min/year.
6. eGFR <30 ml/min/1.73m^2

It is recognized that CKD in elderly population is progressively increasing and lack of adequate studies in this population cohort leads to scarcity of data. Hence adequate studies and trials are required addressing CKD in the elderly.

MATERIAL

AND

METHODS

The study was conducted in Institute of Internal Medicine, Rajiv Gandhi Government General Hospital, Chennai - 600003 between April 2015 and September 2015. Patients presenting to the Emergency department in Institute of Internal Medicine and Nephrology Ward were considered for the study. Rajiv Gandhi Government General Hospital is a tertiary care referral center in Chennai catering to the referral services from hospitals across Tamil Nadu. Hence the subjects in the study represented various population from across Tamil Nadu

NUMBER OF SUBJECTS:

100

INCLUSION CRITERIA:

1. Patients with Chronic Kidney Disease as defined by National Kidney Foundation (NKF) Kidney Disease: Improving Global Outcome (KDIGO) 2012 clinical practice guidelines
2. Patients with Age > 60 years

EXCLSUION CRITERIA

1. Patients with Chronic Kidney disease aged less than 60 years

Patients presenting to the emergency department in the institute of internal medicine serially and patients admitted in the nephrology ward were subjected to the history and physical examination according to the questionnaire and the investigations done were recorded in the proforma The following parameters were assessed

1. History was obtained according to the proforma. History of diabetes mellitus, systemic hypertension, smoking, alcohol consumption, other substance abuse, duration of CKD were noted down
2. The body mass index, vital signs of the patient, ambulant status of the patient, systemic findings, cognitive dysfunction as assessed by MMSE and mini cog test, fundus examination to look for diabetic retinopathy and hypertensive retinopathy, clinical hearing assessment, presence of joint disease were assessed.
3. The frequency of malnutrition as the stage of CKD progressed and the average of Body mass index across different stages of CKD were analyzed.
4. The presence of other geriatric complications including frailty as assessed by osteoporotic fracture index, falls risk assessment, urinary incontinence was also assessed
5. The investigations including hemoglobin, mean corpuscular volume, liver function test, Urine routine, renal function testing including the

presenting creatinine and creatinine from the previous medical records, serum sodium, serum potassium, arterial pH, pCO2, bicarbonate, Anion gap and special investigation if any done were noted down.

6. The hemoglobin distribution and the prevalence of anemia in different stages of CKD, the type of anemia (microcytic, normocytic) were assessed in the study population.
7. The distribution of serum sodium and potassium in different stages of CKD and the prevalence of hyponatremia and hyperkalemia in different stage CKD were analysed. Comparison of potassium level between diabetics and non diabetics were also assessed.
8. The distribution of the arterial pH and arterial bicarbonate in different stages were assessed.
9. The eGFR was calculated using the variables required using cockloft gault formula, MDRD formula and CKD - EPI formula.
10. The ultrasonography and the ejection fraction if clinically warranted were done and noted. The mean distribution of the ejection fraction in the subjects assessed were analyzed. The distribution of mild, moderate and severe Left ventricular systolic function as assessed by echocardiography were analyzed.
11. The complications including Acute on chronic kidney disease, Acute pulmonary edema, acidosis, hyperkalemia, encephalopathy,

pericarditis were recorded and assessed for correlation with the progression in the stage of CKD

12. Viral Markers including HbsAg, antiHCV, HIV were recorded
13. The Renal replacement therapy if initiated including mode, centre, funding for renal replacement therapy were recorded
14. The probable etiology of the CKD were ascertained and their distribution frequency in the study population were analyzed
15. The patient were followed up either by contacting through telephone or during their review visit. If the patient is not on follow up the reason for not being in follow up were noted down.
16. The analysis of statistical significance were carried out using SPSS software version 20.0

CONSENT:

Written and informed consent were obtained from all the participants in feasible cases or their attenders

ETHICAL COMMITTEE APPROVAL:

The study was approved by INSTITUE OF ETHICAL COMMITTEE of MADRAS MEDICAL COLLEGE

CONFLICT OF INTEREST:

NONE

SPONSORSHIP:

NONE

RESULTS

The results were tabulated and analyzed. The demographic distribution, clinical profile, complications, the distribution of complications across the stages of CKD according to the eGFR as estimated by CKD - EPI formula, etiology and follow up of the patients were tabulated and analyzed.

The subjects included in the study were more than 60 years. The minimum age of the subject was 60 years and the maximum age of the study population was 80 years. The mean age of the population studied was 65.2 years with a standard deviation of 5.1. The distribution of the curve is depicted as follows

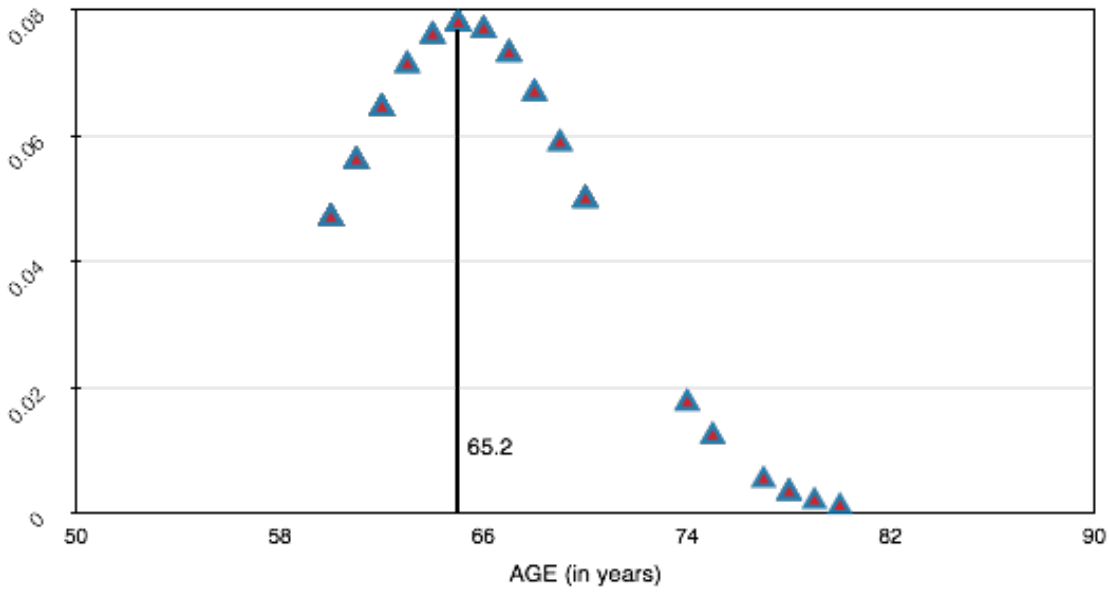

Fig 1. Distribution of Age of the study population (Mean of 65.2 ± 10.2)

The study population included 46 males and 54 female.

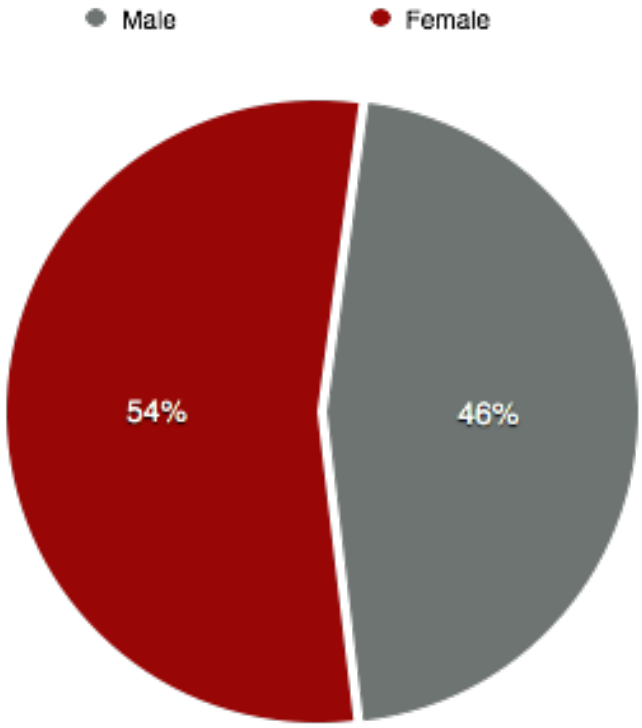

Fig 2. Pie chart depicting the Gender distribution in the population

The study population consisted of 61 patients with diabetes mellitus amounting to 61% of the study population and 75 subjects with systemic hypertension amounting to 76 percent of the study population. 38 patients with Diabetes mellitus had non proliferative diabetic retinopathy and 1 patient had proliferative diabetic retinopathy. 17 patients with hypertension had hypertensive retinopathy.

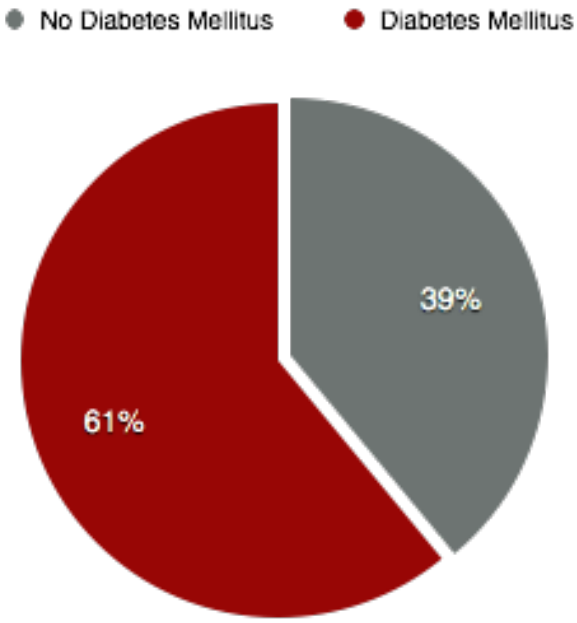

Fig 3. Pie Chart showing 61% of patients with diabetes mellitus and 39% without diabetes mellitus

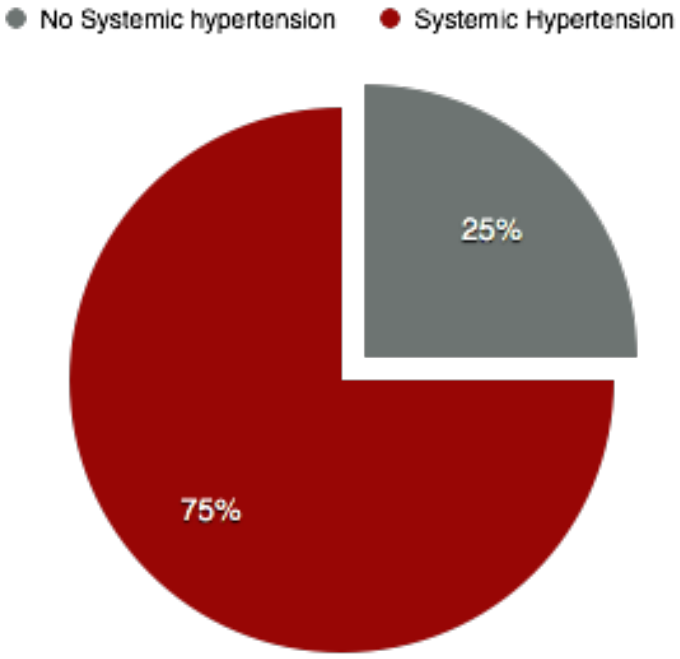

Fig 4. Pie Chart showing 75% patients with hypertension and 25% patients without hypertension.

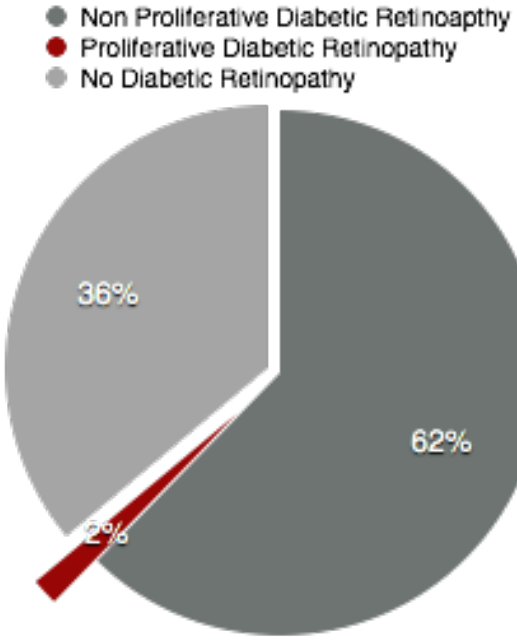

Fig 5. Pie Chart showing the prevalence of diabetic retinopathy. 62% of diabetics had non proliferative diabetic retinopathy and 2% of the diabetics had proliferative diabetic retinopathy

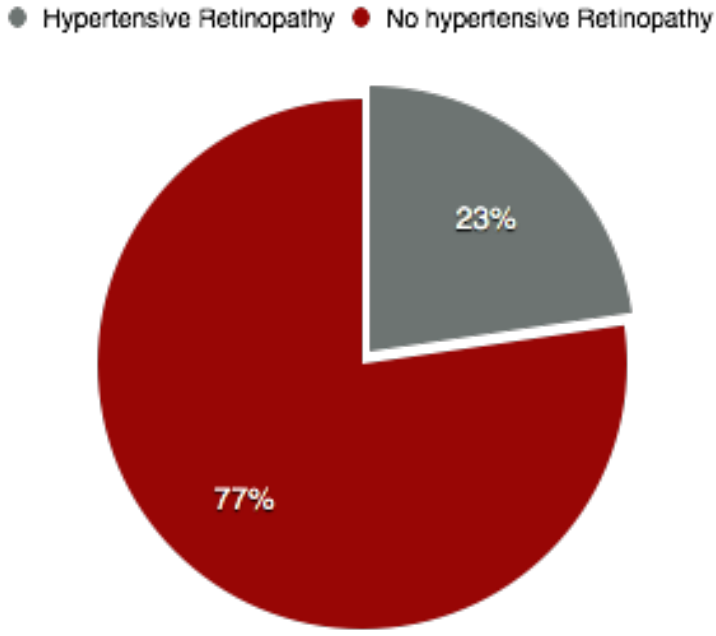

Fig 6. Pie Chart showing the prevalence of hypertensive retinopathy. 23% of the hypertensive subjects had hypertensive retinopathy.

25 subjects among males consume alcohol accounting for 54.3% of the male population and 1 subject among the female population consume alcohol accounting for 1.8% of the female population

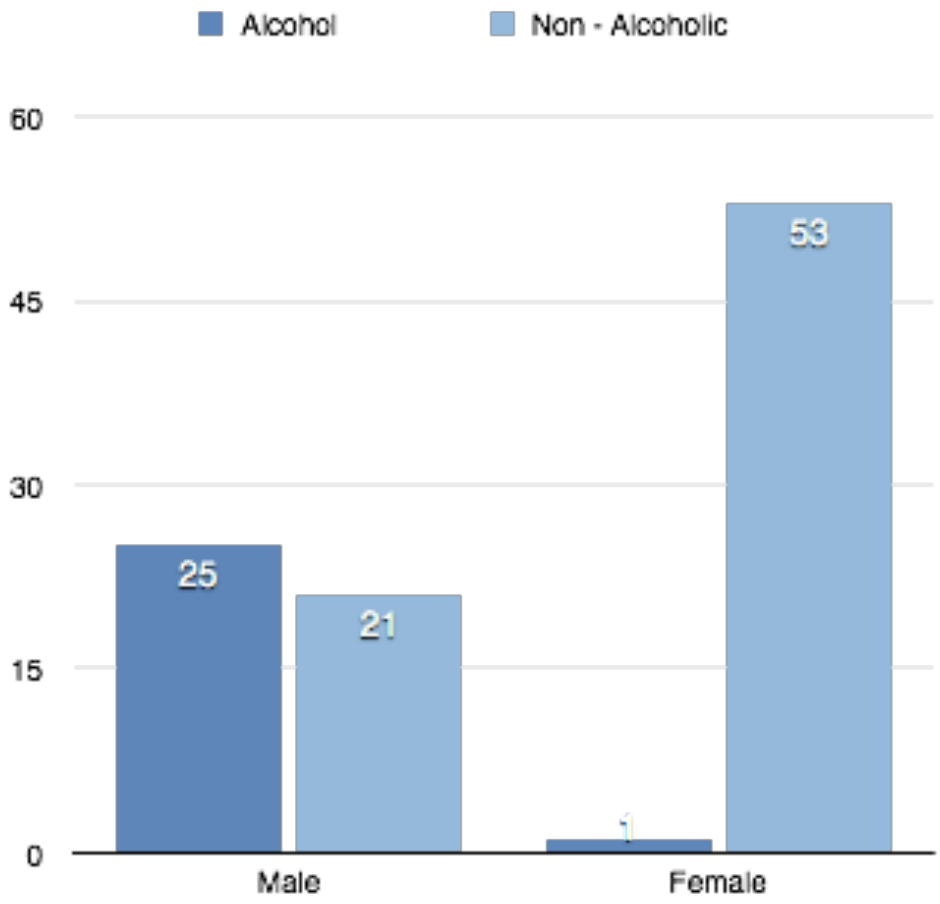

Fig 7. Bar diagrams depicting the consumption of alcohol across gender.

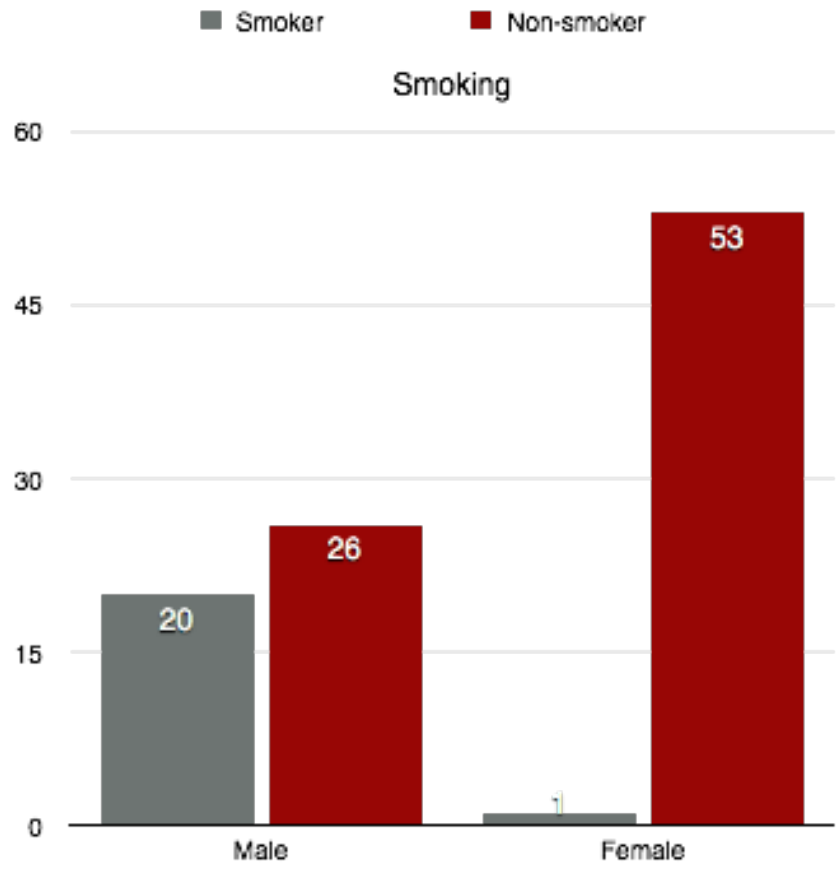

Fig 8. Bar diagrams depicting the smoking pattern in the study population

20 patients among males smoke cigarettes accounting for 43.5% of the male population and 1 patient among females smoke accounting for 1.8% of female population.

The study population had the duration of CKD distributed over a range from minimum 3 months to a maximum of 14 years. The mean of the duration of CKD was 1.49 with a standard deviation of 1.9. The distribution curve of the duration of CKD of the study population is as shown below.

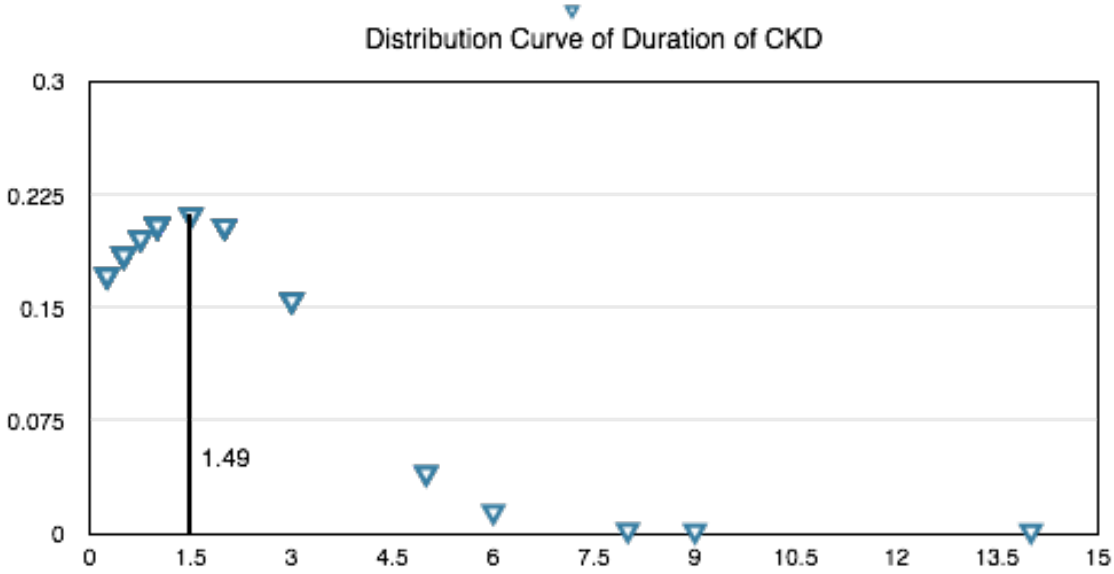

Fig 9. Distribution curve of Duration of CKD of the study population with a mean of 1.49 ± 3.8 (2SD).

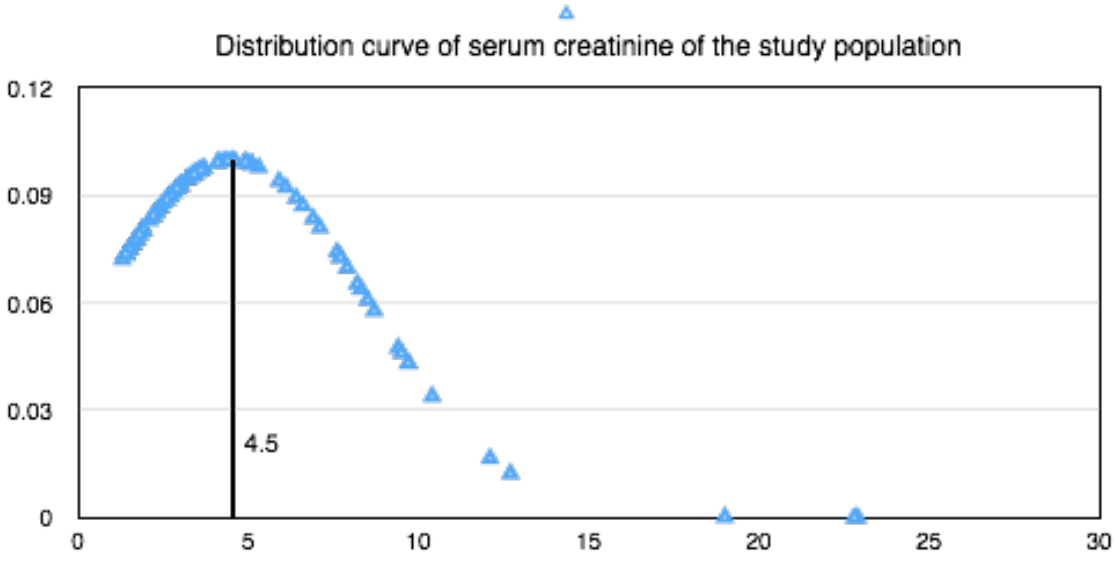

Fig 10. Distribution Curve of serum creatinine. Mean 4.5 ± 8.0 (2SD)

The presentation serum creatinine of the study subjects were documented and the eGFR was calculated as per the CKD - EPI equation. The mean presenting creatinine of the population was 4.5 mg/dl with a standard deviation of 4.0 mg/dl.

The study population were categorized according to the stage of CKD and the distribution of the subjects across various stages of CKD were analyzed. The patients presented in various stages of CKD ranging from IIIa, IIIb, IV and V. The frequency of the study population across various groups is depicted as follows

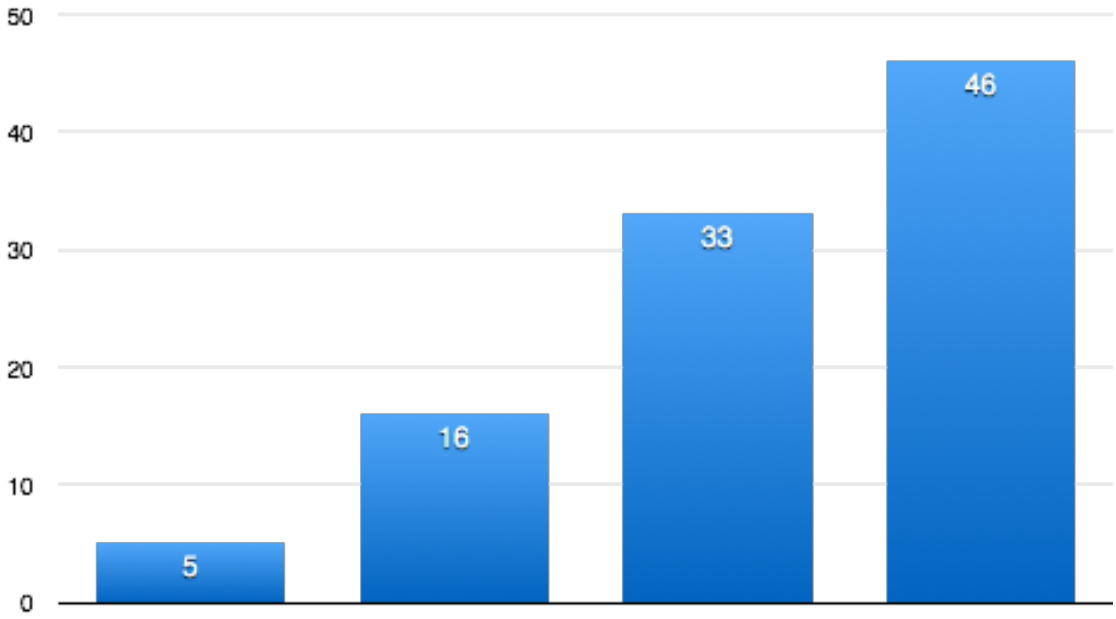

Fig 11. Bar Graph showing distribution of the study population across various stages of CKD as classified by NKF - KDIGO guidelines according to eGFR.

The analysis of the prevalence of the complications such as cognitive dysfunction, frailty, urinary incontinence, falls risk and ambulant state of the patients in the geriatric population were assessed.

The cognitive dysfunction as assessed by Mini-mental state examination concluded that 17 patients had cognitive dysfunction and 65 patients did not have cognitive dysfunction. The cognitive dysfunction as assessed by Mini-cog test concluded that 15 patients had cognitive dysfunction and 67 patients did not have cognitive dysfunction. 18 subjects could not be assessed for cognitive dysfunction using either MMSE or Mini-Cog test because of the presence of encephalopathy. Though the cognitive dysfunction as assessed by MMSE estimated 2 patients to have cognitive dysfunction in addition to that estimated by mini cog test, it was not statistically significant.

In the patient population studied, 13% had urinary incontinence, 17% were underweight and malnourished. 60% of the patient population were ambulant at presentation. 40% of study population were either bed ridden or required support for mobility.

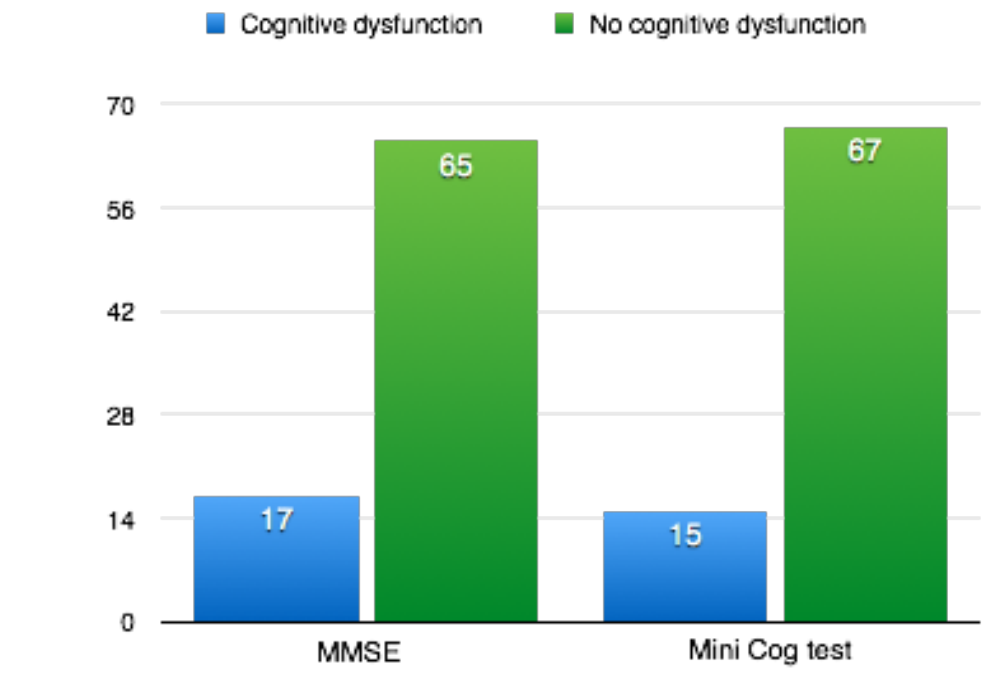

Fig 12. Bar Graphs showing cognitive dysfunction as assessed by MMSE and Mini cog test (p value 0.693518 - not significant)*

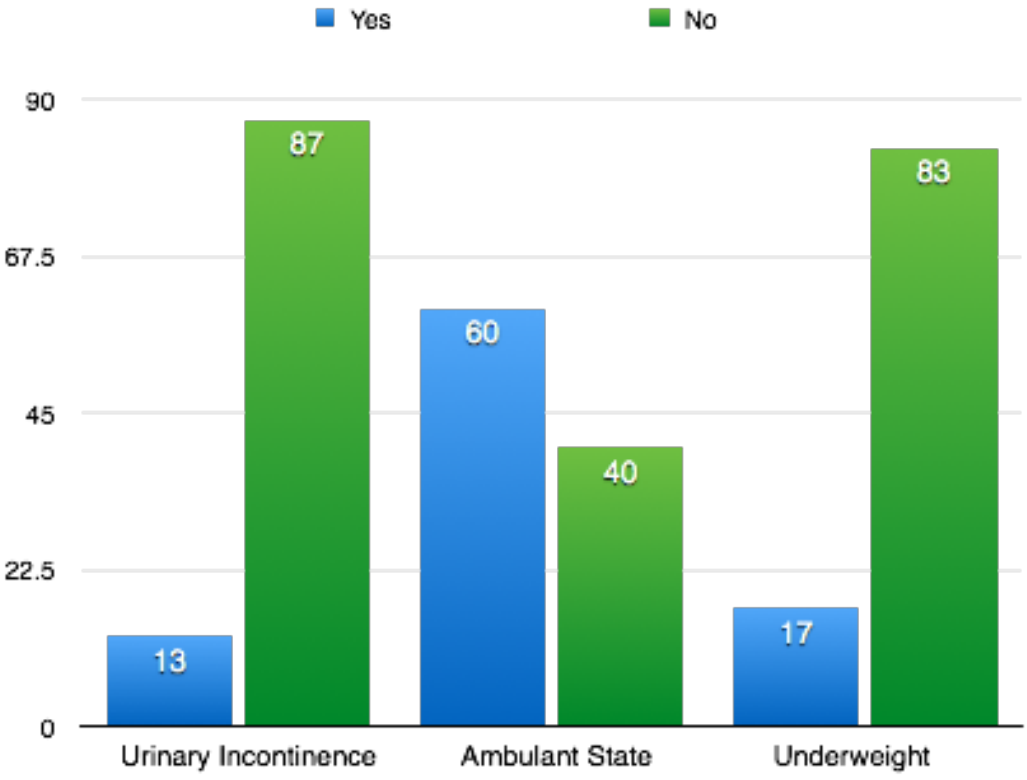

Fig 13. Bar Graphs showing prevalence of Urinary incontinence, ambulant state, Underweight in the study population

In the study population 65% of the patients had low risk for fall, however 4% and 31% had moderate and high risk for fall respectively.

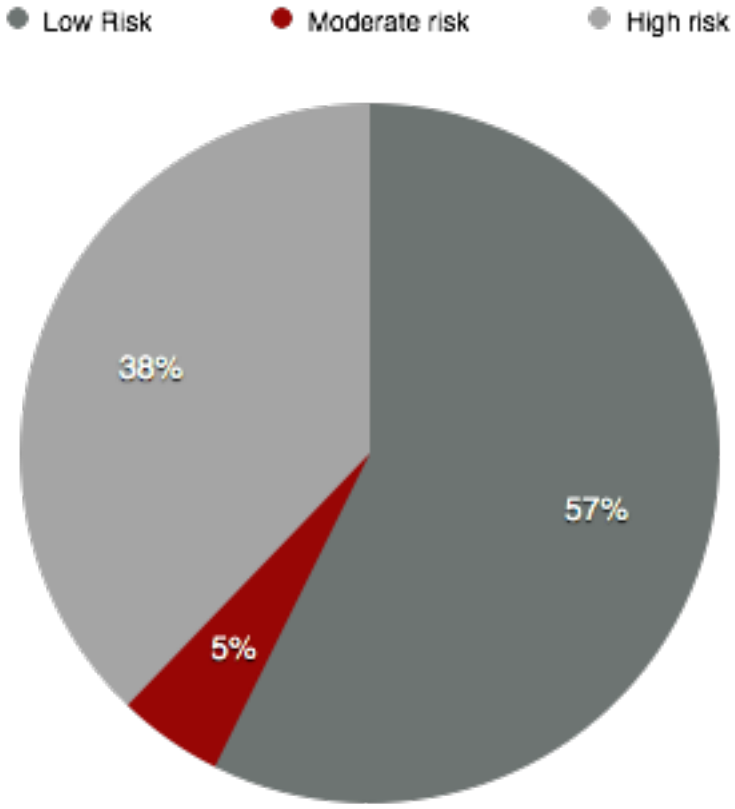

Fig 14. Pie Chart showing the risk of fall in the study population

In the study population, 45% of the patients had degenerative joint disease involving the knee joint, hip joint or shoulder joint as evidenced by clinical examination or radiological imaging. 2% of the patients had fractures involving the neck of femur due to a fall. 53% of the population did not have any joint involvement. The Frailty assessment by Osteoporotic fracture index revealed than 76% of the study population was frail, 6% pre-frail and 18% without any frailty.

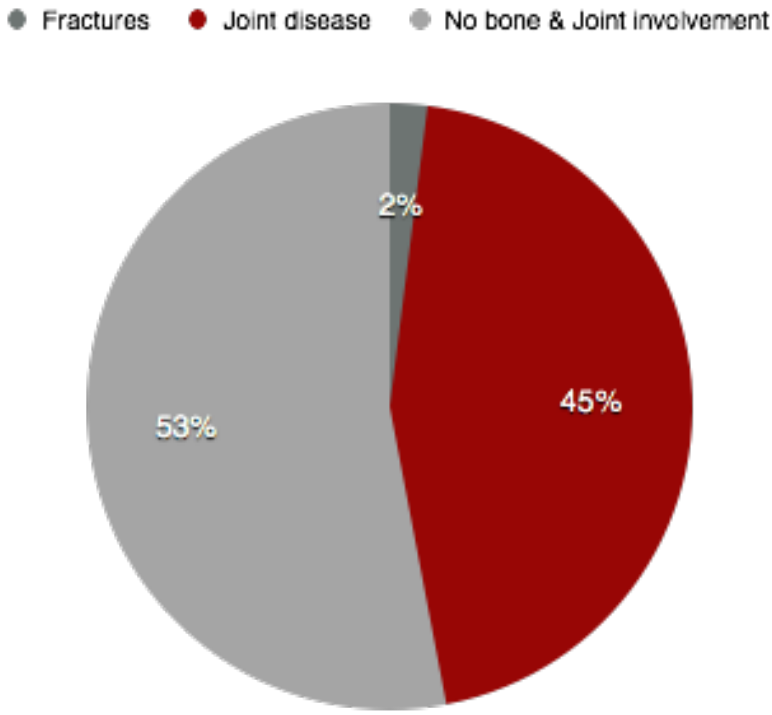

Fig 15. Pie chart showing joint involvement and fractures in the study population

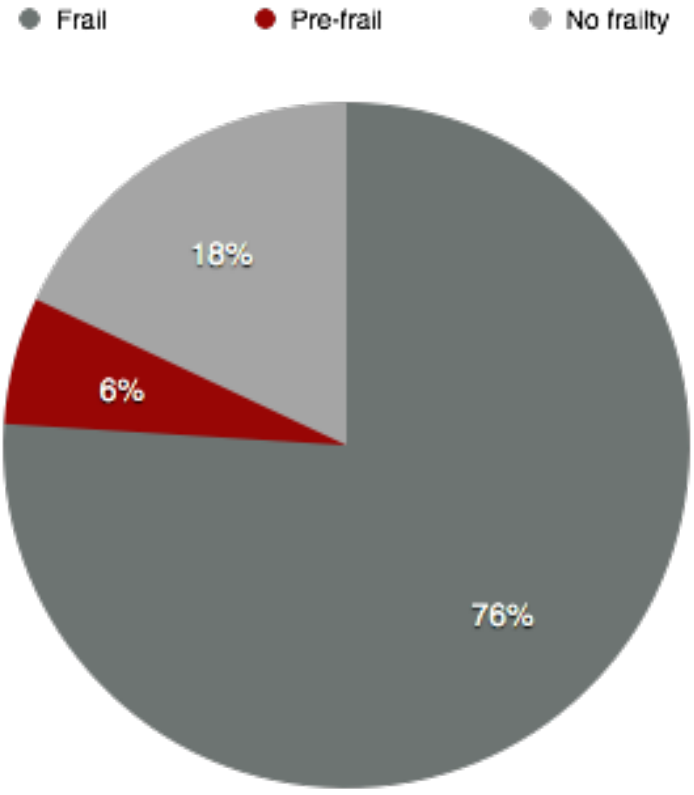

Fig 16. Pie chart showing frailty assessment

Though the absolute number of subjects involved by malnutrition and cognitive dysfunction is more in advanced stages of CKD, the percentage of the corresponding population being involved is not statistically significant. However, the quantitative analysis of the Body Mass Index across the stages of CKD had shown the mean of BMI in stages IIIa, IIIb, IV, V were 23.58, 23.24, 21.17, 21.16. The difference in BMI between the stages were statistically significant.

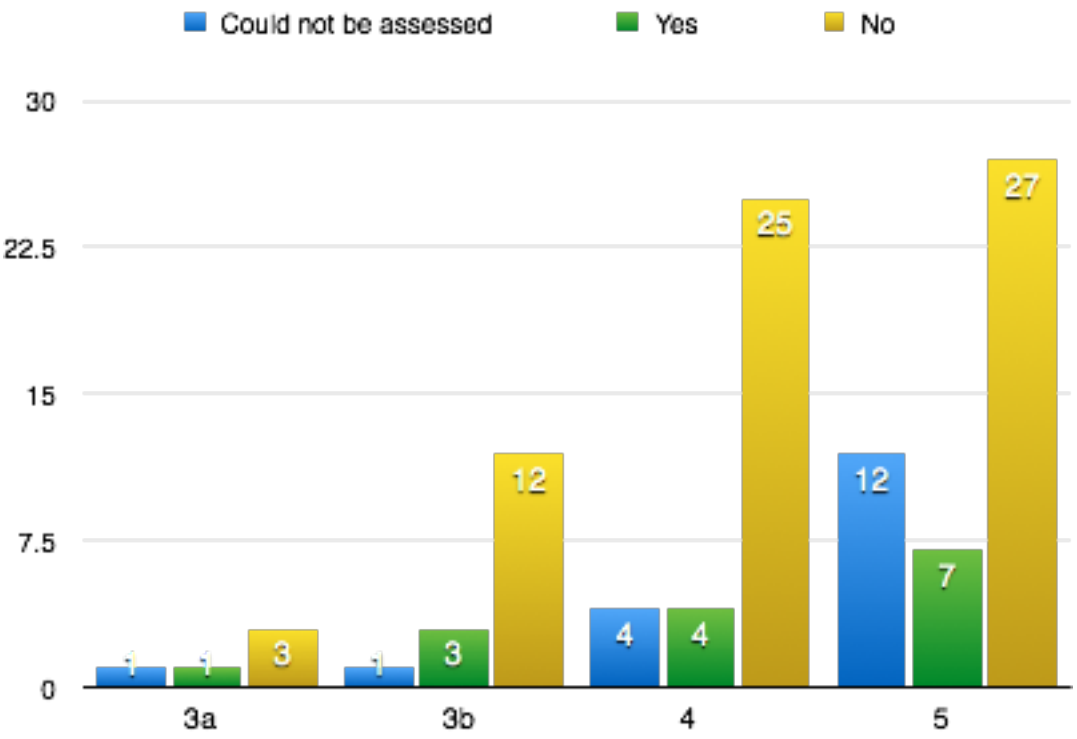

*Fig 17. Bar Graph showing frequency of cognitive dysfunction in patients with different stages of CKD. *p value 0.544 - not significant*

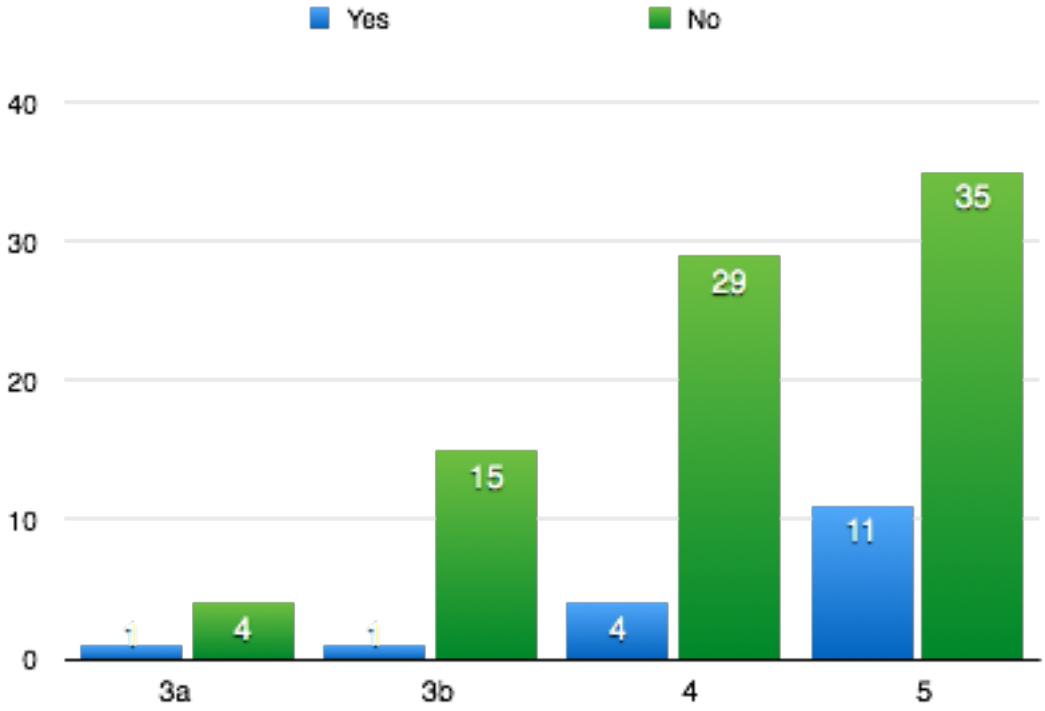

*Fig 18. Bar Graph showing frequency of malnutrition in patients with different stages of CKD. *p value 0.326 - not significant*

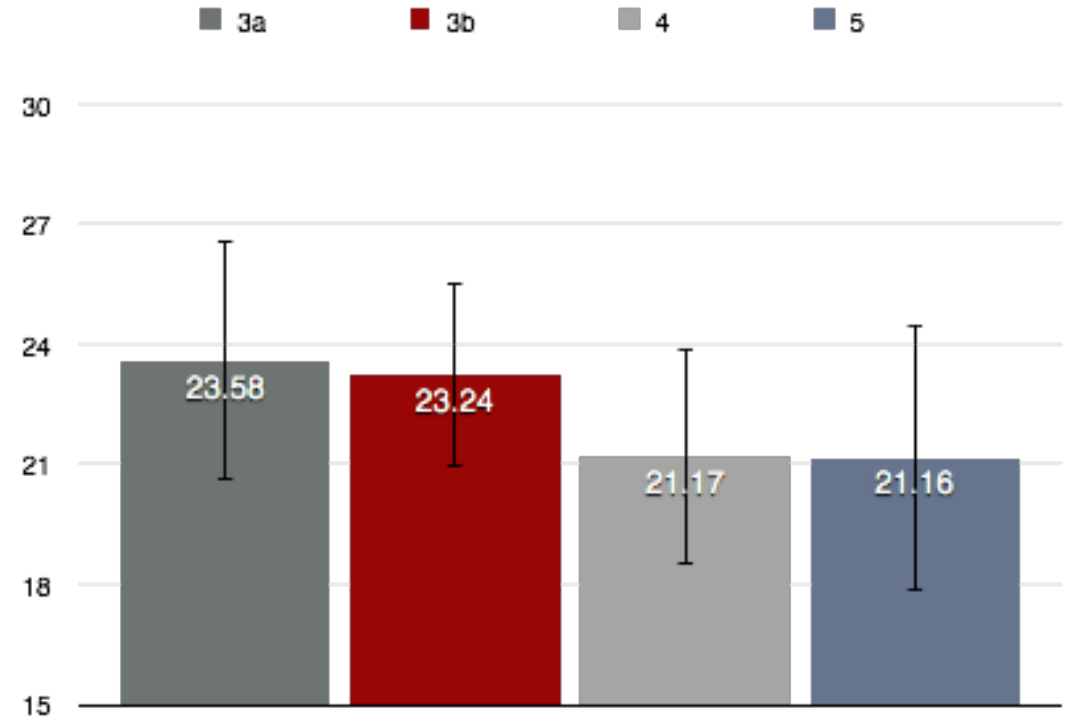

*Fig 19. Bar Graph showing comparison of means of BMI in different stages of CKD. * p value 0.036 - significant (p < 0.05)*

The frequency of various complications such as Acute on chronic kidney disease, acute pulmonary edema, acidosis, pericarditis and other complications were analysed and compared with different stages of CKD. The increased frequency of complications such as acute on chronic kidney disease and acidosis as the stage of CKD progressed were statistically significant. Though the absolute number of patients involved had increased as the stage of CKD progressed, it was not statistically significant with respect to acute pulmonary edema, encephalopathy and other complications. The other complications that had occurred in the study population includes 4 patients with hypokalemia, 2 patients with cerebrovascular accident and 1 patient with hypoglycemic episode. None of the patients had complications of pericarditis.

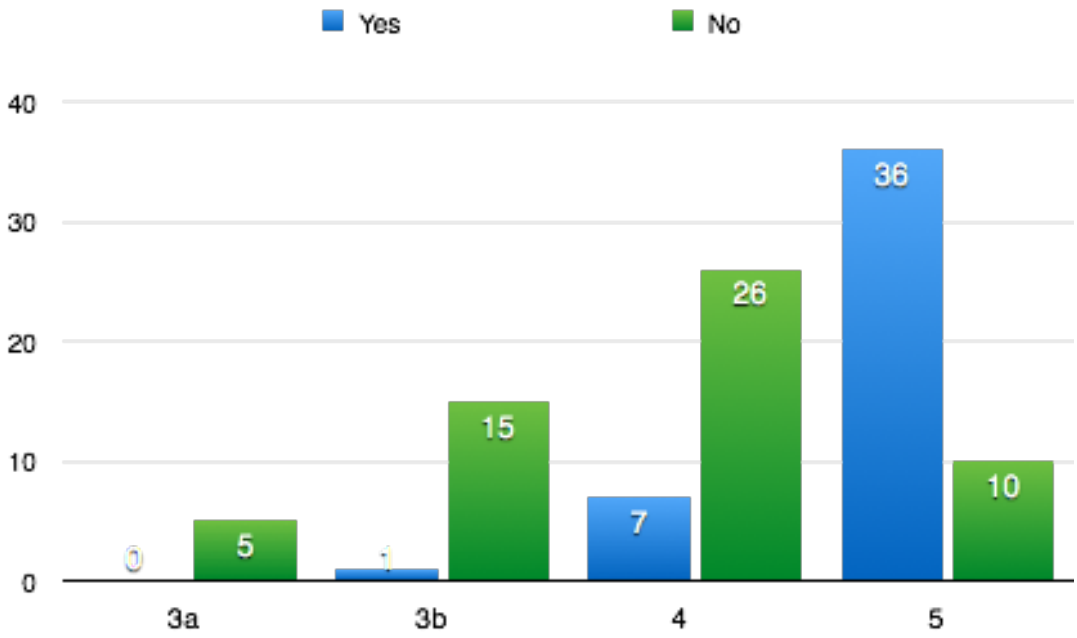

*Fig 20. Bar Graph showing frequency of Acute on CKD in patients with different stages of CKD. *p value <0.001 - significant*

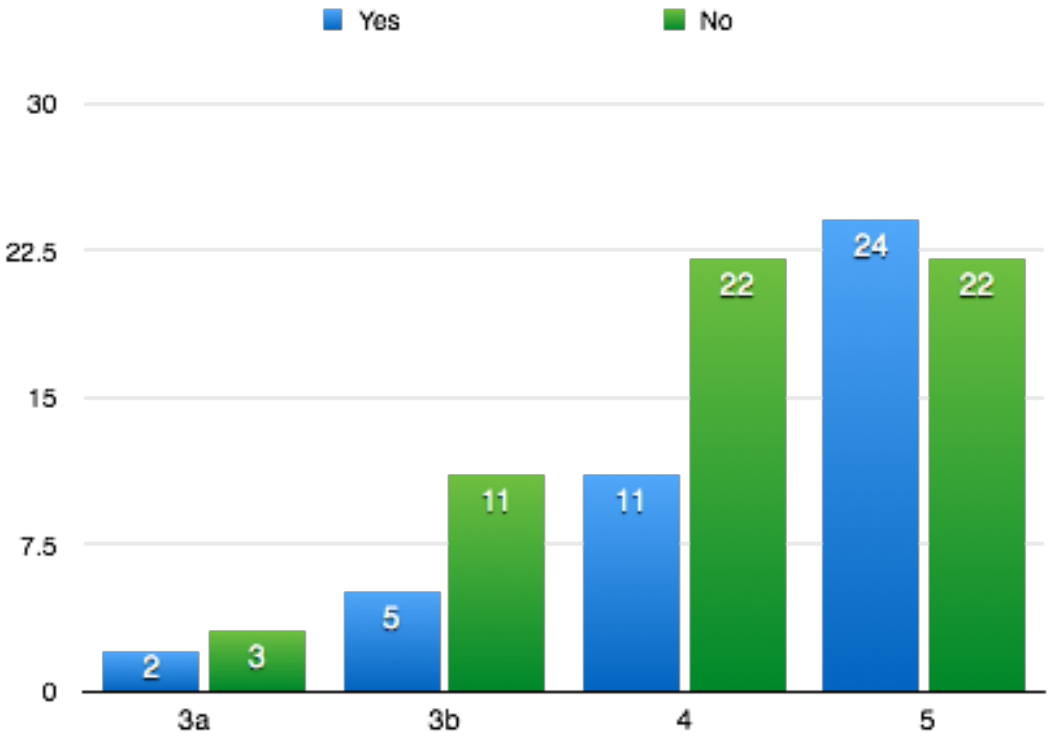

*Fig 21. Bar Graph showing frequency of Acute pulmonary oedema in patients with different stages of CKD. *p value 0.291 - not significant*

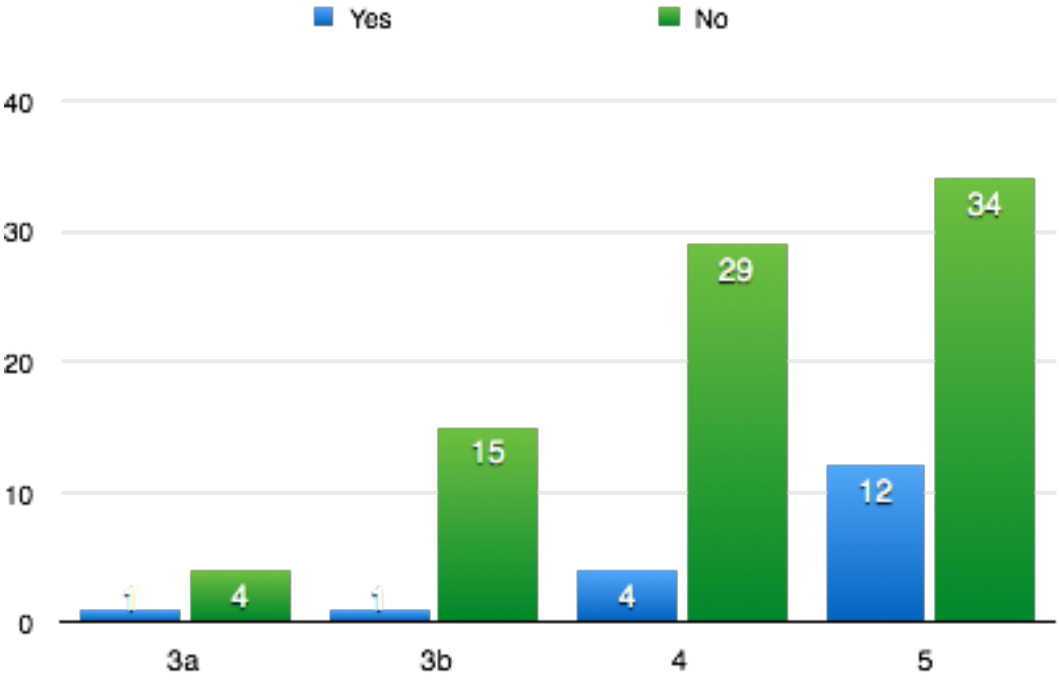

*Fig 22. Bar Graph showing frequency of Encephalopathy in patients with different stages of CKD. *p value 0.229 - not significant*

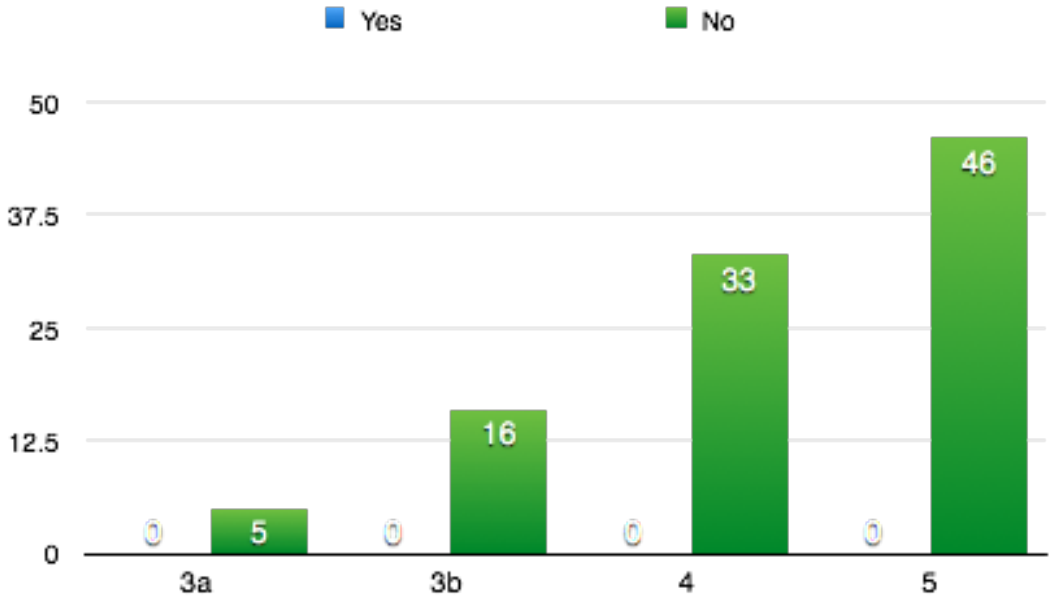

Fig 23. Bar Graph showing frequency of Pericarditis in patients with different stages of CKD.

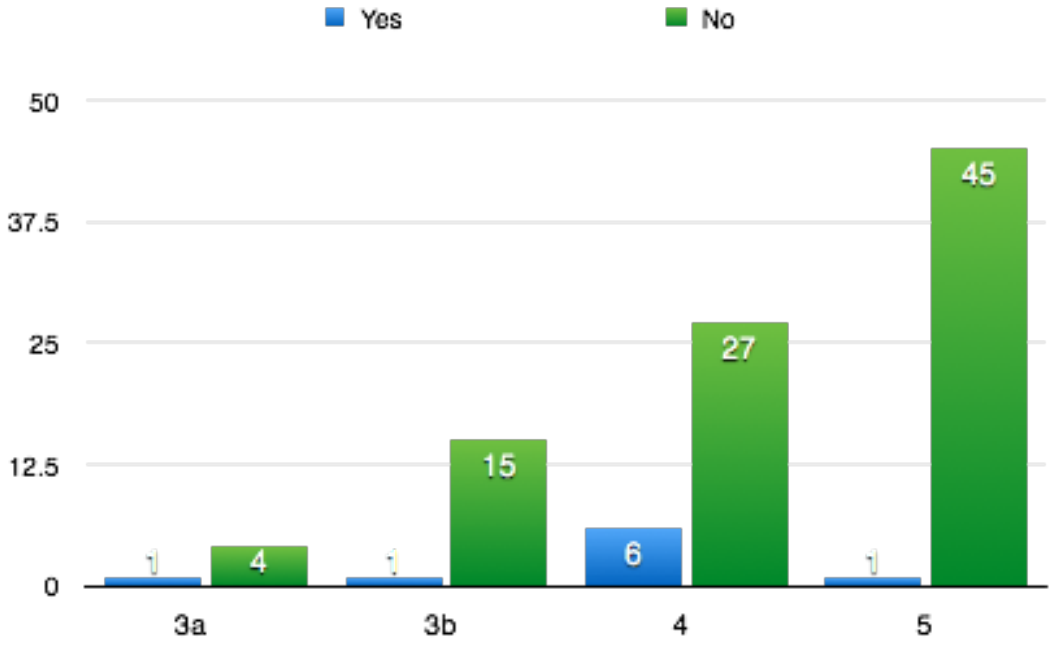

*Fig 24. Bar Graph showing frequency of Other complications in patients with different stages of CKD. *p value - 0.075 - not significant*

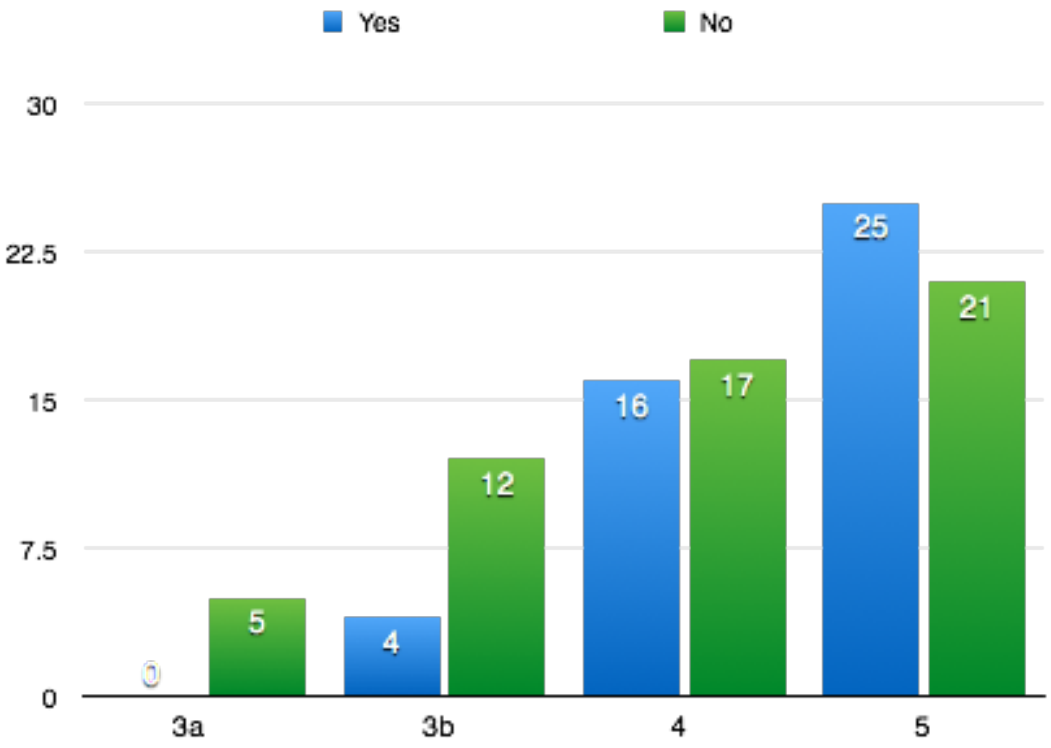

*Fig 25. Bar Graph showing frequency of acidosis in patients with different stages of CKD.*p value - 0.037 - significant at p <0.05*

However quantitative analysis of the mean of the arterial pH across various stages of CKD has shown a statistically significant difference in pH between groups. The quantitative analysis of the mean of the bicarbonate levels in various stages of CKD has shown a statistical significant difference

The mean sodium level between different stages of CKD were not statistically significant. Neither was the prevalence of hyponatremia statistically significant between different stages of CKD. The quantitative analysis of the mean of potassium levels has shown statistically significant difference between different stages of CKD. And the

frequency of hyperkalemia has shown statistically significant difference between different stages of CKD.

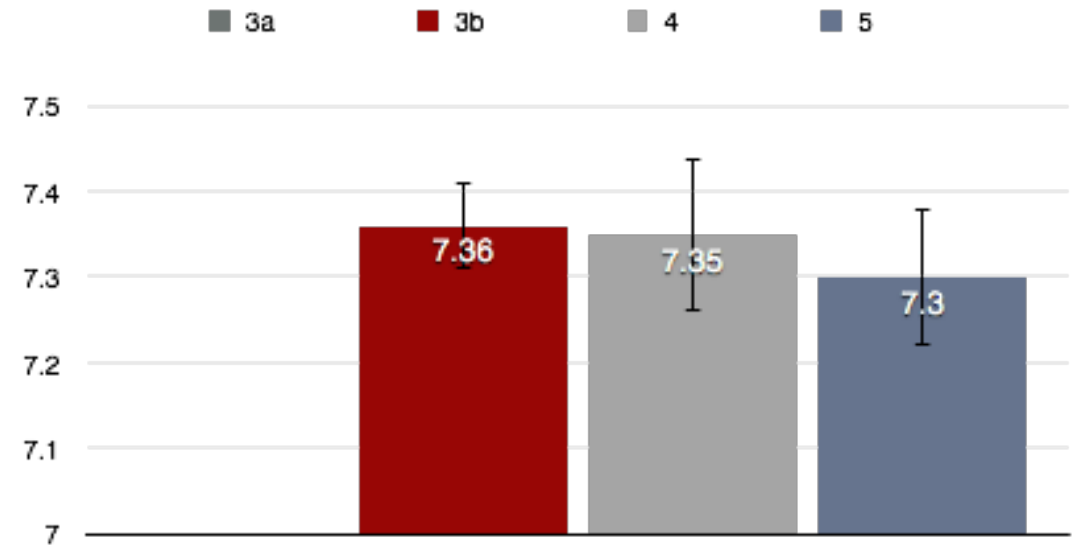

*Fig 26. Bar Graph showing comparison of mean of arterial pH in different stages of CKD. * p value 0.045 - significant at p < 0.05*

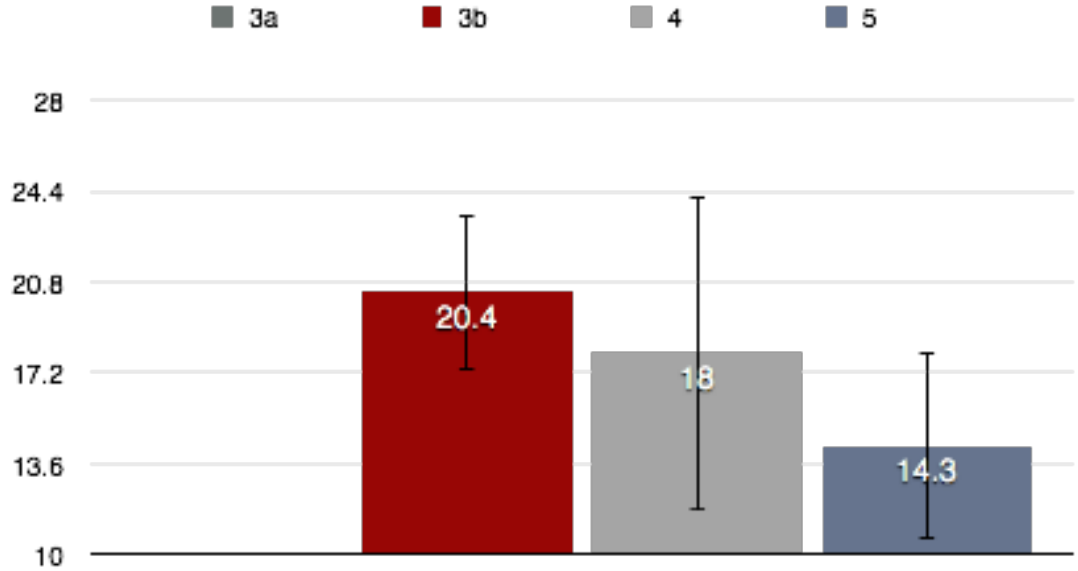

*Fig 27. Bar Graph showing comparison of mean of serum bicarbonate in different stages of CKD. * p value 0.0008 - significant at p < 0.001*

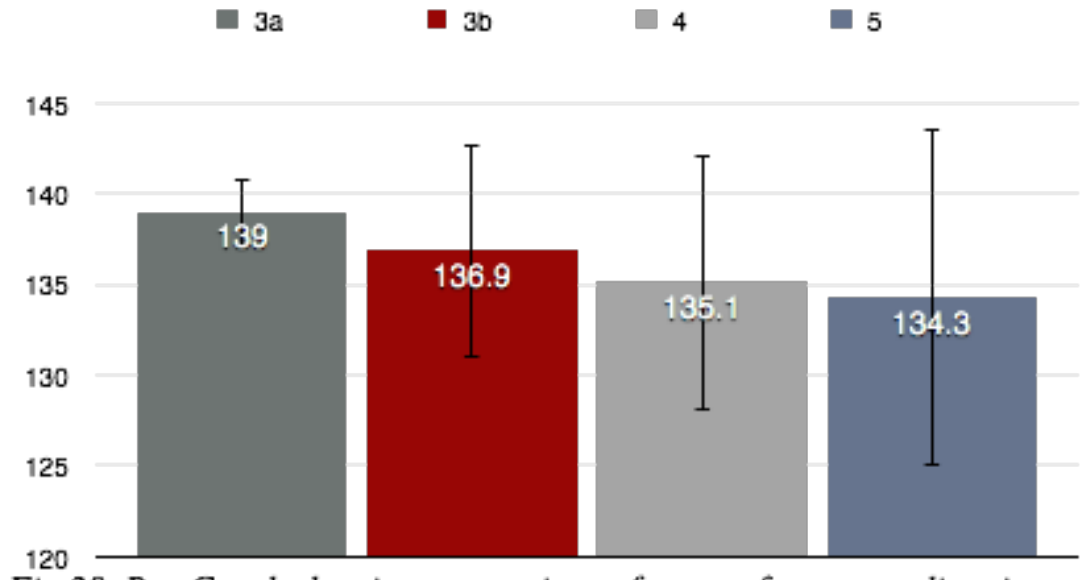

*Fig 28. Bar Graph showing comparison of mean of serum sodium in different stages of CKD. * p value 0.508 - not significant.*

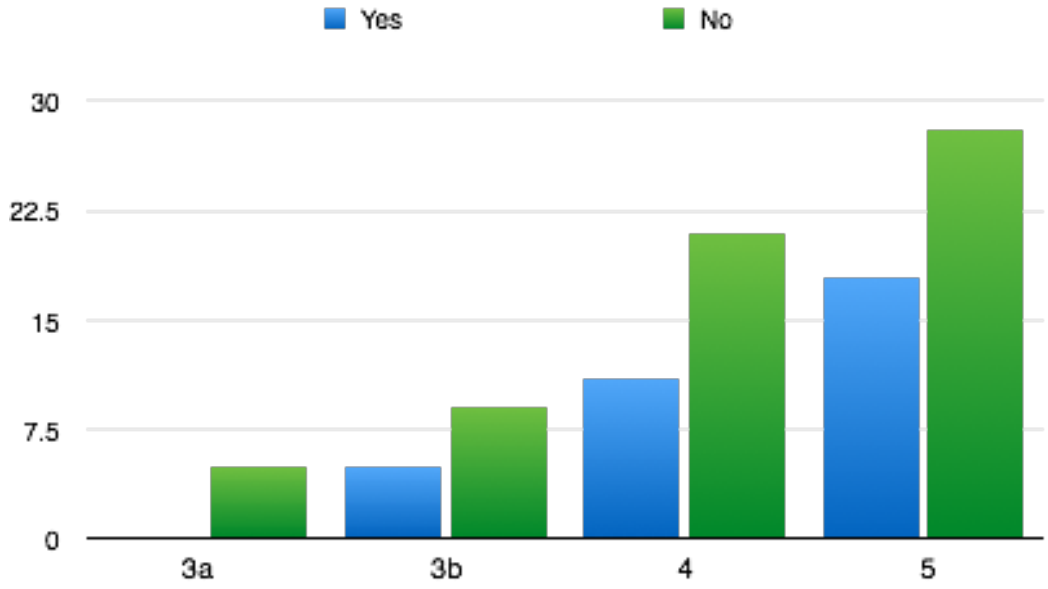

*Fig 29. Bar Graph showing frequency of hyponatremia in patients with different stages of CKD.*p value - 0.38- not significant*

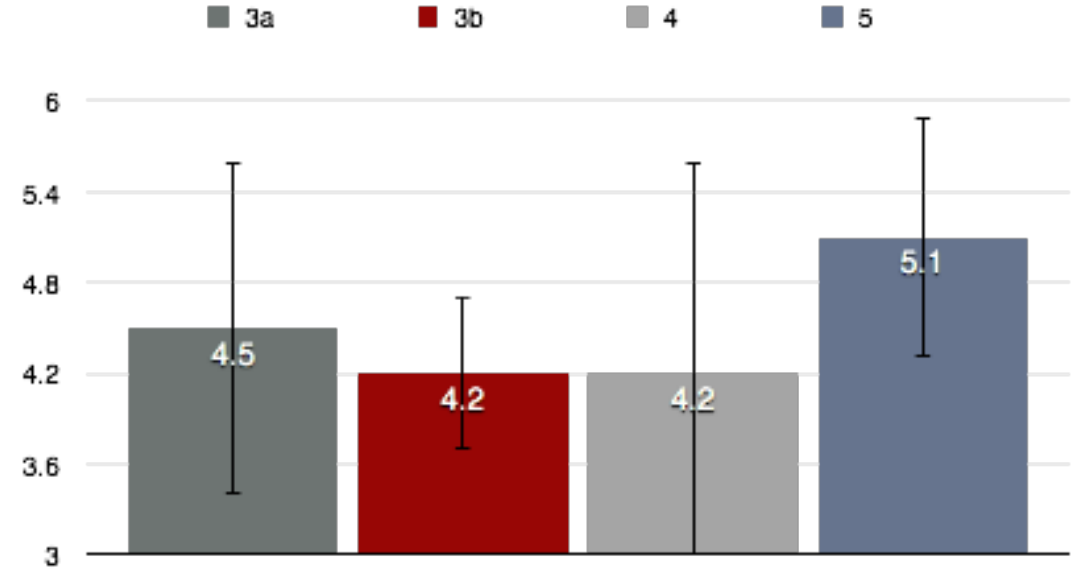

*Fig 30. Bar Graph showing comparison of mean of serum potassium in different stages of CKD. * p value 0.016 - significant at p < 0.05.*

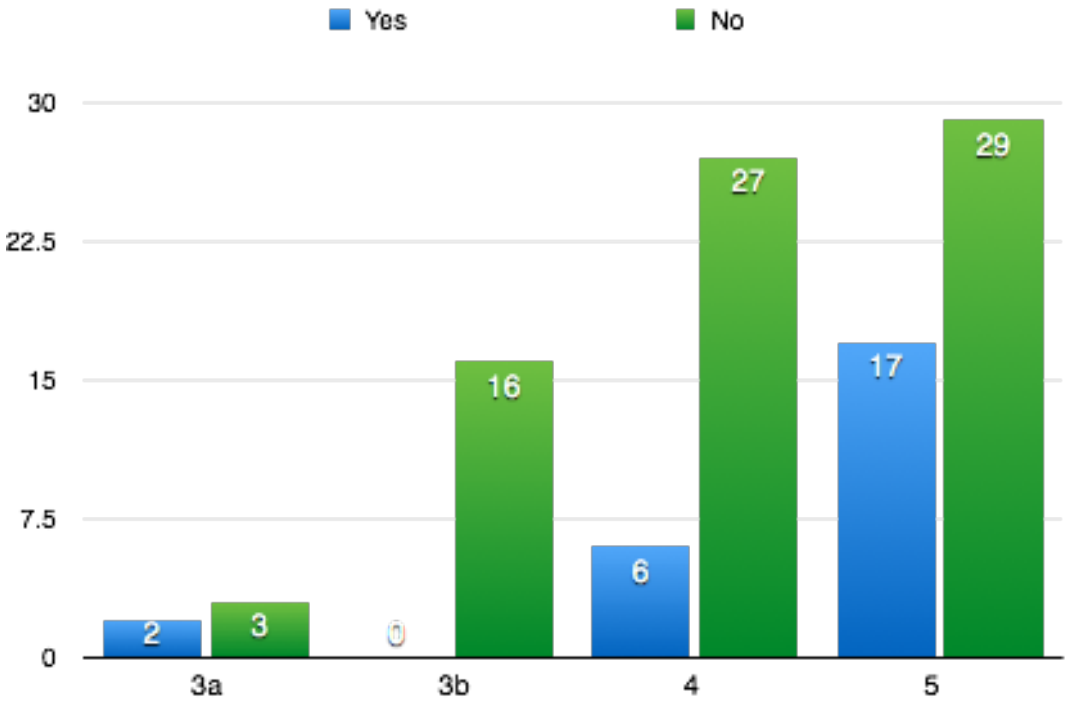

*Fig 31. Bar Graph showing frequency of Hyperkalemia in patients with different stages of CKD. *p value 0.015 - significant (p < 0.05)*

There was no statistically significant difference in the frequency of hyperkalemia between diabetics and non diabetics. Neither was there any statistically significant difference in the mean of potassium between the 2 groups.

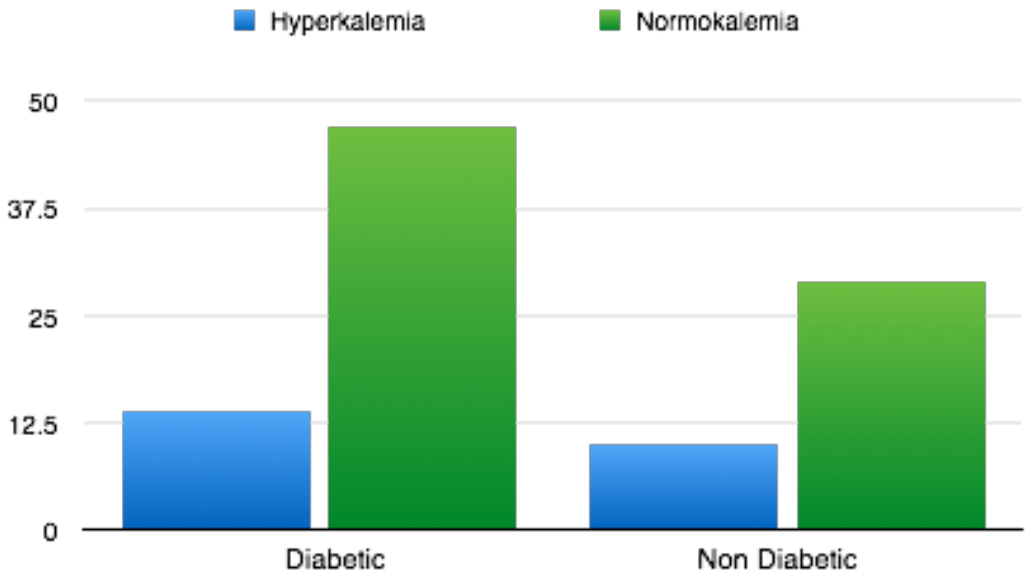

*Fig 32. Bar Graph showing frequency of Hyperkalemia in diabetics and non diabetics. *p value 0.75 - not significant*

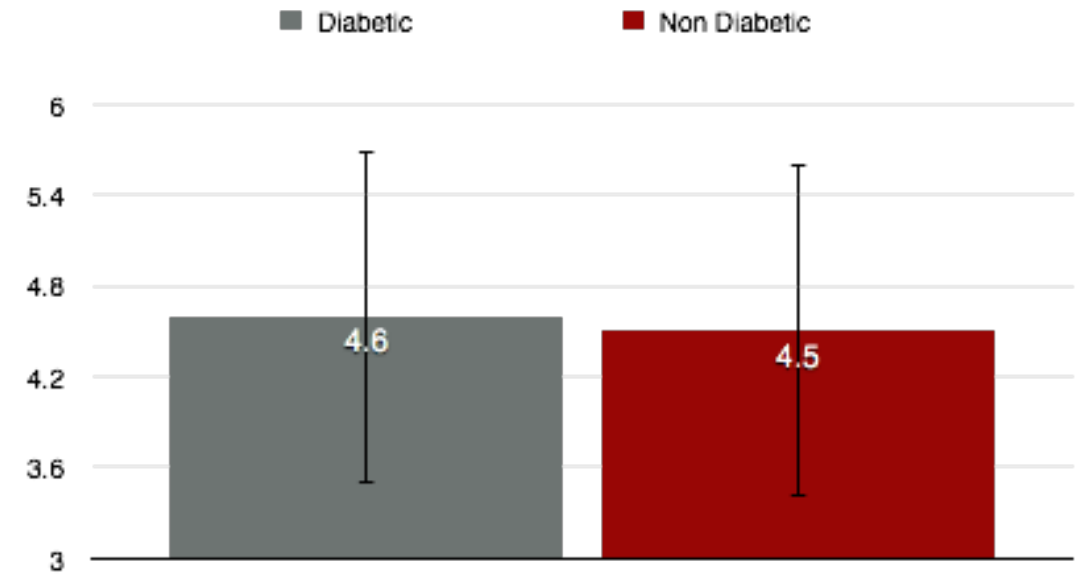

*Fig 33. Bar Graph showing comparison of mean of serum potassium in diabetics and non diabetics. * p value 0.016 - significant at p < 0.05.*

The mean distribution of hemoglobin in the study population was 9.2 g/dl with a standard deviation of 2.2. The normal distribution curve of the population is as shown below

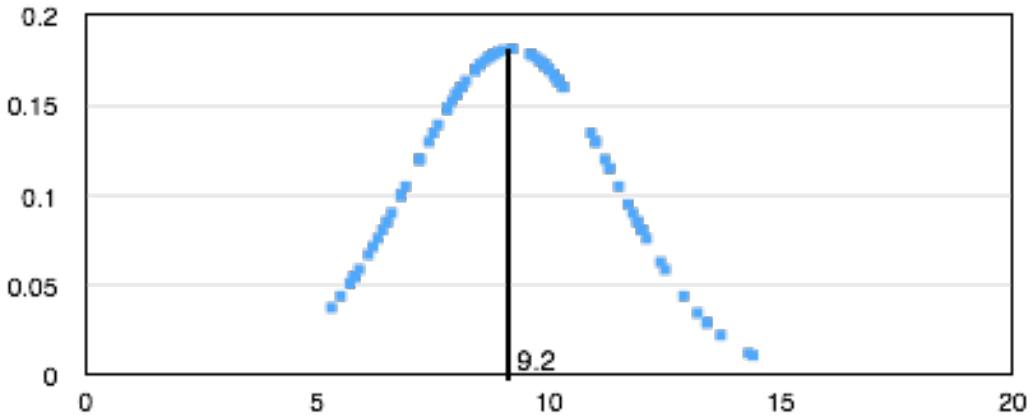

Fig 34. Distribution Curve of Hemoglobin mean 9.2 ± 4.4 (2SD)

The prevalence of anemia in the study population was 91% with 93% of males 89% of females were affected. 70% of the patients had normocytic anemia and 30% of the patients had microcytic anemia.

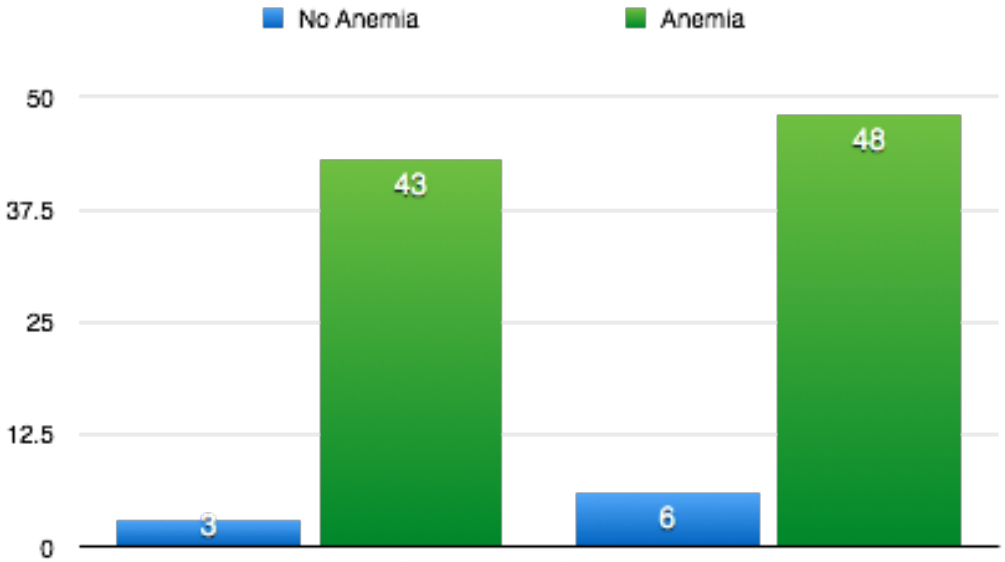

Fig 35. Bar Graph showing frequency of Anaemia in Male and Female patients.

The comparison of the mean of hemoglobin between the stages of CKD showed a statistical significance between the groups.

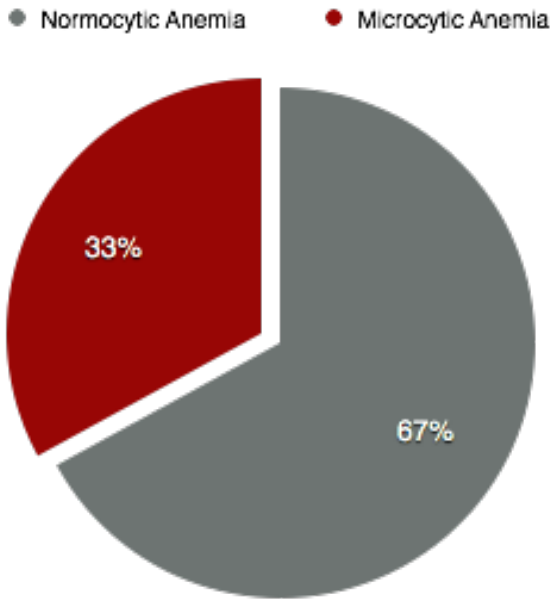

Fig 36. Pie chart showing percentage of patients with normocytic and microcytic anaemia

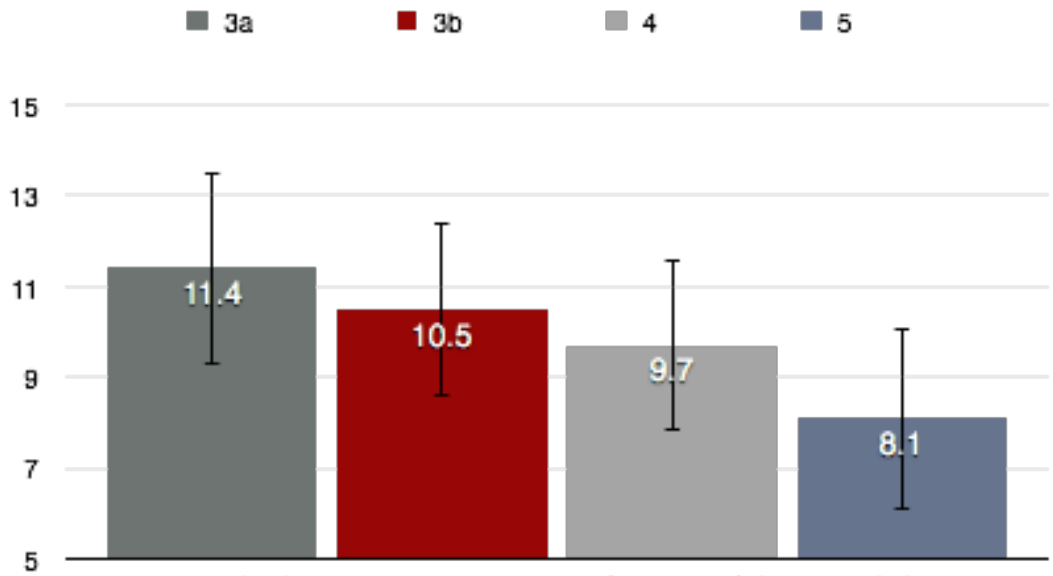

*Fig 37. Bar Graph showing comparison of mean of haemoglobin in different stages of CKD. * p value 0.016 - significant at p < 0.001.*

The distribution of the liver enzymes including SGOT, SGPT and alkaline phosphatase were analyzed. The mean of SGOT was 24.5 with a standard distribution of 18.3. The mean of SGPT was 24.1 with a standard deviation of 17.5. The mean of alkaline phosphatase was 107.5 with a standard distribution of 68.4

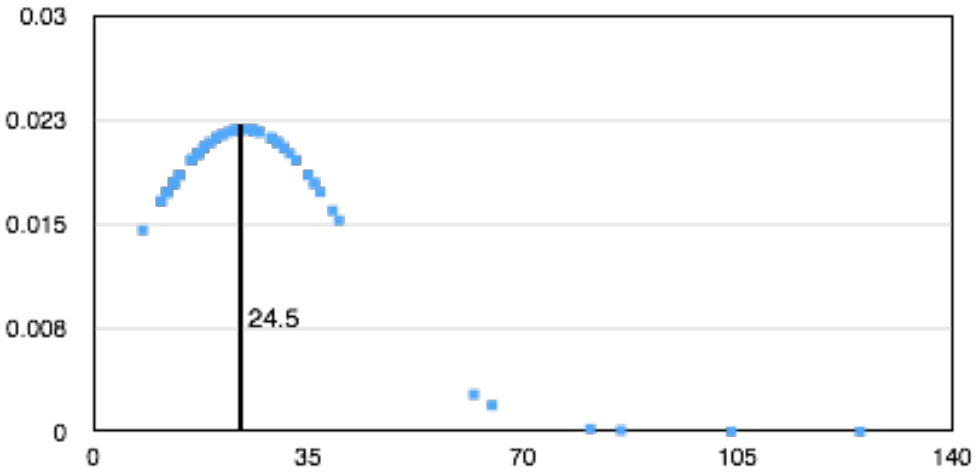

Fig 38. Distribution Curve of Aspartate Transaminase. Mean 24.5 ± 36.6 (2SD)

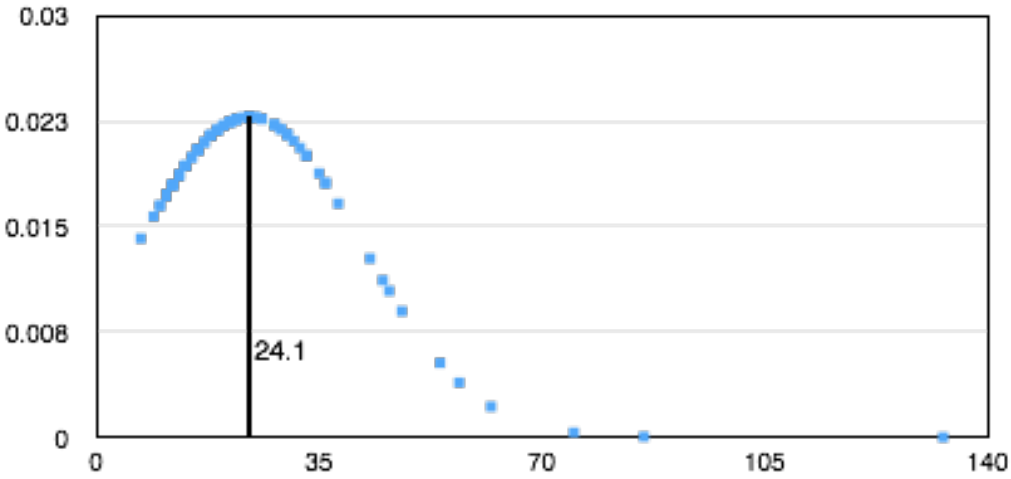

Fig 39. Distribution Curve of Alanine Transaminase. Mean 24.1 ± 35.0 (2SD)

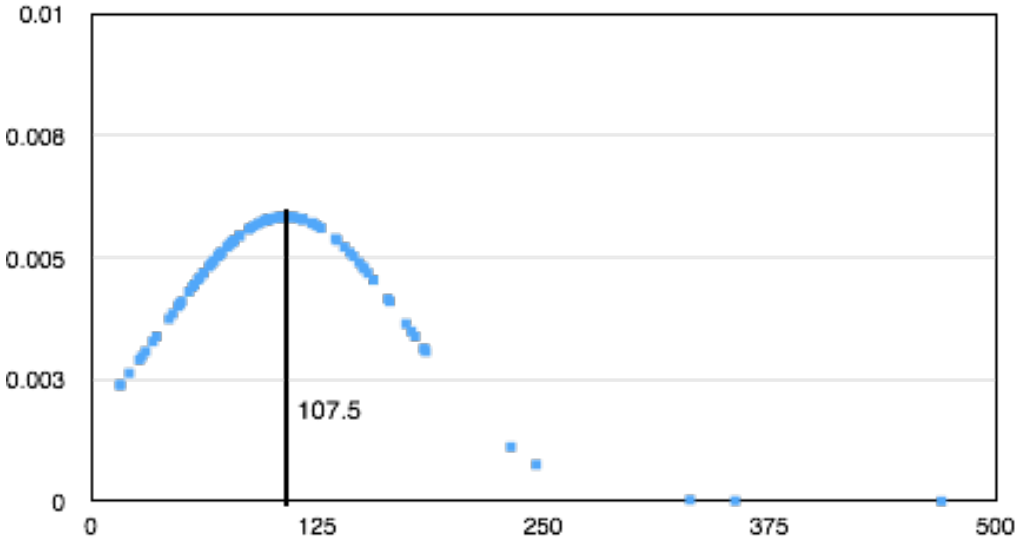

Fig 40. Distribution Curve of Alkaline Phosphatase. Mean 107.5 ± 136.8 (2SD)

The Left ventricular ejection fraction was estimated in 54 subjects. The distribution curve of the ejection fraction of the study population is as shown below with a mean of 53.5 with a standard deviation of 11.0. The frequency of ejection fraction

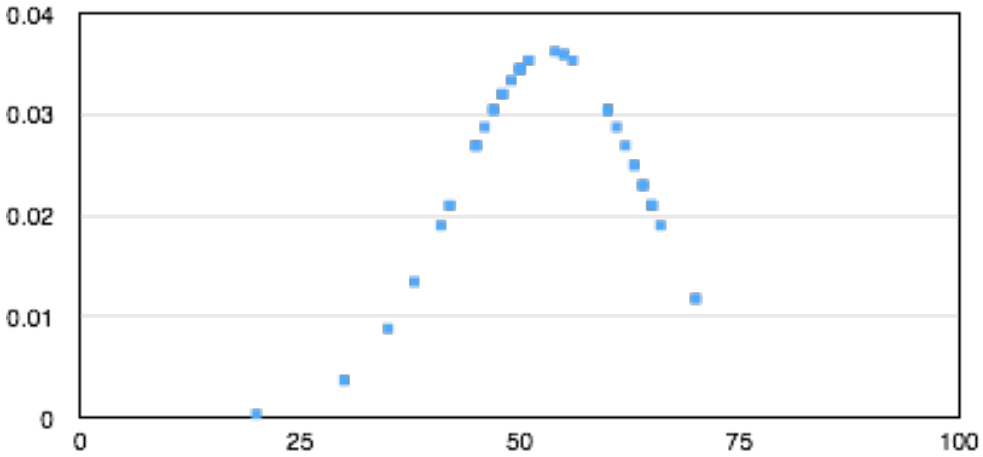

Fig 41. Distribution Curve of LV ejection fraction. Mean 53.5 ± 22.0 (2SD)

The prevalence of Left ventricular systolic dysfunction in the subjects who were assessed were 53.7%. The frequency of patients with mild, moderate and severe Left ventricular systolic dysfunction were 21, 4 and 4 respectively.

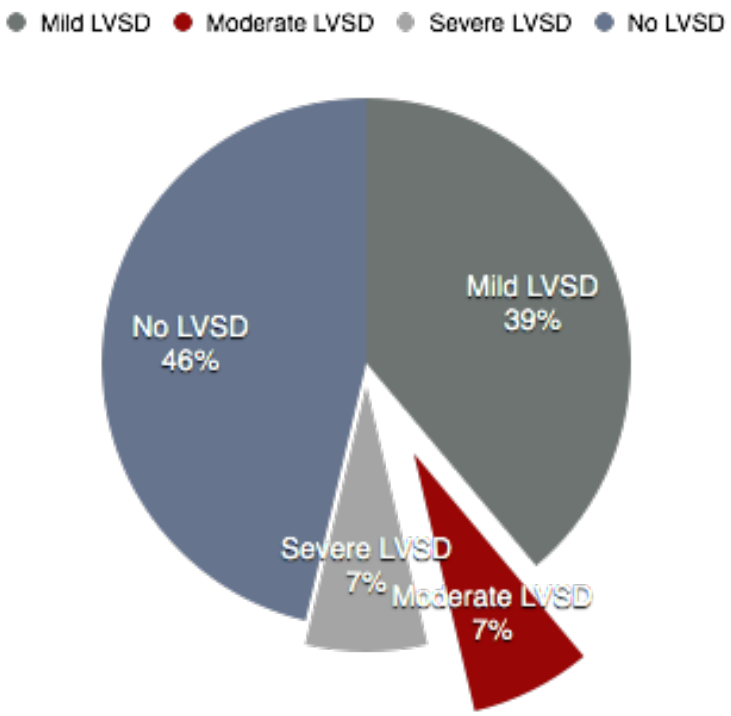

Fig 42. Pie chart showing percentage of patients on PD, HD and PD / HD

Analysis of the patients on renal replacement therapy revealed that a total of 25% of the study population were on renal replacement therapy. 2% of the population were already on hemodialysis, 3% of the population were on peritoneal dialysis and converted to hemodialysis and 20% of the patients had been initiated on peritoneal dialysis. None of the patients were initiated for renal transplantation.

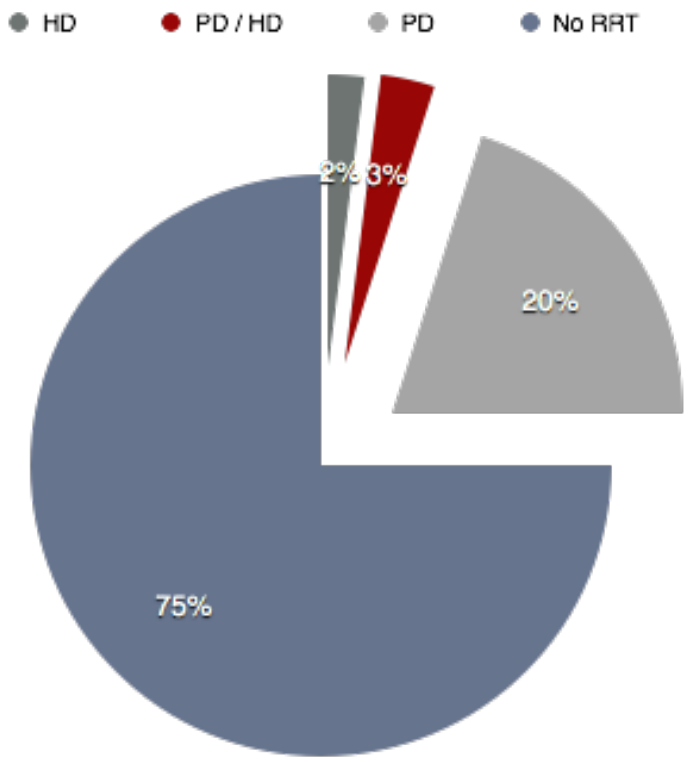

Fig 43. Pie chart showing percentage of patients on PD, HD and PD / HD

The contribution of the various etiology and contributory factors in the development of chronic kidney disease in the study population were analyzed. The frequency of Diabetes Mellitus, Systemic hypertension, Chronic glomerulonephritis, Chronic interstitial nephritis, Ischemic nephropathy, Autosomal dominant polycystic kidney disease, myeloma kidney was 61, 75, 19, 8, 3, 2, 1 respectively. However, the cause could not be ascertained in 4 of the patients.

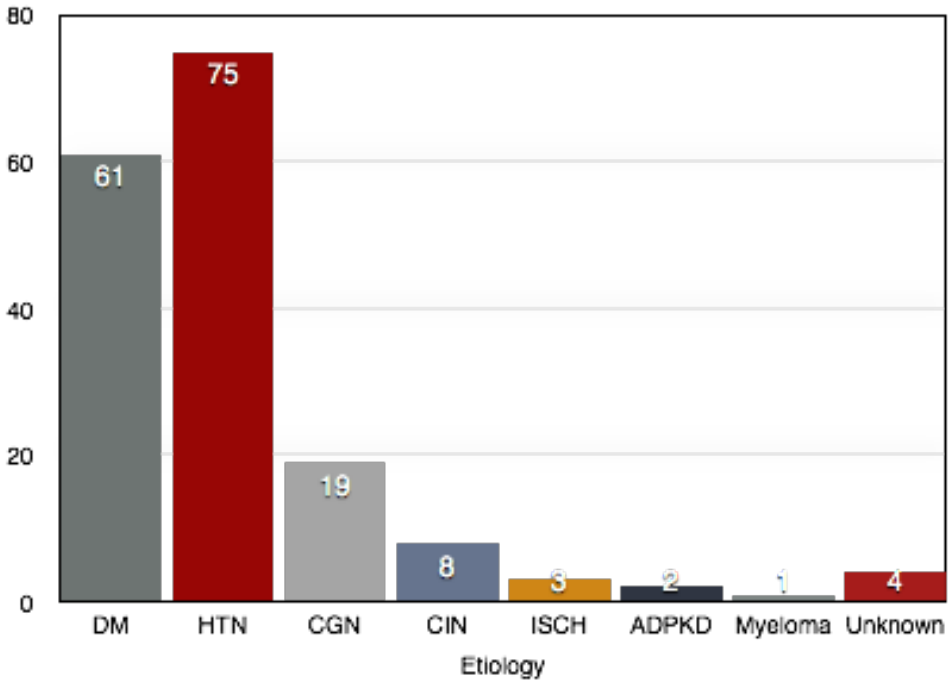

Fig 44. Bar Graph showing frequency of aetiology and contributory factors in the development of CKD

Subsequent follow up of the patient were analysed based on if they are residing in urban area or rural area. In the study population, 57% of the patients were from urban area and 43% of the patients were from rural area. 22 patients from the urban population (38.6%) and 17 patients from the rural population (39.5%) could not be contacted. The loss of contact was either dus to loss of communication or due to death of the subject.

Among the patients who had been contacted, 6 patients from the urban population (17.14%) and 15 patients from the rural population (57.6%) did not follow up for medical consultation. This difference between the rural and the urban population was statistically significant.

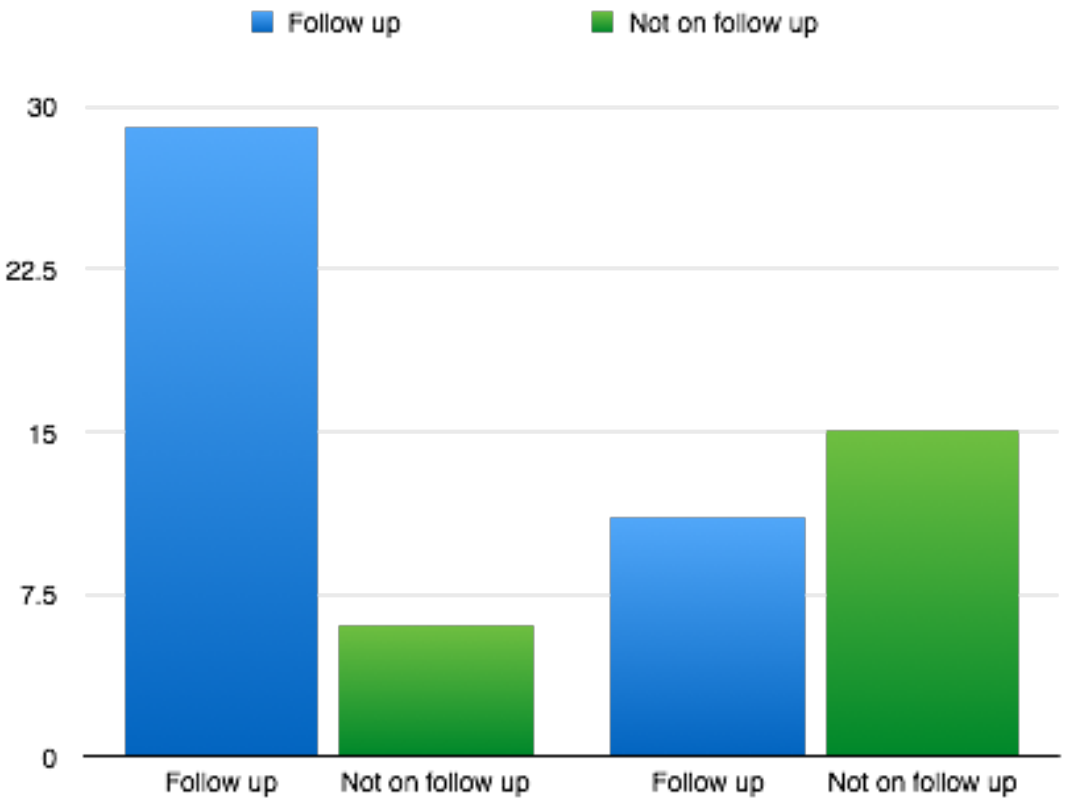

*Fig 45. Bar Graph showing frequency of urban and rural patients on follow up.*p value - 0.0009 - significant at p<0.001*

DISCUSSION

The above analysis of etiology, clinical and lab profile and the outcome of the patient with chronic kidney disease has wreathed into the wider aspects of chronic kidney disease in geriatric population. The above analysis addresses both the complications of CKD and the complications pertaining to geriatric population. This analysis encompassing wider aspects of the chronic kidney disease in geriatric population arose to the need created by the definition of CKD as defined by the NKF KDOQI that was introduced in the year 2002. In doing so, there was a paradigm shift in the perspective of chronic kidney disease. It transformed from treating end stage renal disease to preventing and retarding the progression of CKD. But in doing so larger proportion of older individuals were addressed under the label, increasing the incidence and prevalence of chronic kidney disease in geriatric population[101]. This further is augmented by the rising geriatric population in the developing low and middle income countries which has resulted from the betterment of life expectancy[97]. It was also noted that it was this geriatric population that was at increased risk of complications from chronic kidney disease[98] and the complication in geriatric population such as cognitive dysfunction, falls and falls risk, frailty[99], urinary incontinence was increased in the geriatric population with CKD than the general geriatric population[100].

The study population had a mean age of 65.2 years with a female

preponderance. The age ranged from a minimum of 60 years to a maximum of 80 years. 54.3% of the male population and 1.8% of the female population consumed alcohol whereas 43.5% of the male population and 1.8% of the female population had history of smoking. None the subjects involved had any history of any other substance abuse. The study population consisted of 61 diabetics and 75 hypertensive patients. The prevalence of diabetic retinopathy was 64% with non proliferative diabetic retinopathy accounting for 62%. This high incidence is explained by the increased association of diabetic retinopathy with overt nephropathy. This finding was similar to the incidence noted by Parvin et al, 1992[102]. Further there was decreased finding of proliferative retinopathy as opposed to the study reported by *Penno et al, 2012*[103] in which proliferative retinopathy was reported in 15.28% of the patients with CKD. This low composition of proliferative diabetic retinopathy can be accounted by the predominant asymptomatic nature of the non proliferative diabetic retinopathy and symptomatic nature of advanced diabetic retinopathy. Hence does not reflect the community prevalence of proliferative and non proliferative retinopathy. The prevalence of hypertensive retinopathy which included Grade 1 and Grade 2 retinopathy accounted for 23% of the patients with hypertension. This is in concordance with the prevalence of retinopathy detected in hypertensive population by Shantha et al, 2010[104].

The study population had predominant patients distributed among the stages of CKD IV and V which accounted 79% of the population. The study population was homogenous in having an overt nephropathy with an estimated GFR of <60 ml/min/1.73m^2. The cognitive function assessed by MMSE had shown 17 patients to be affected by dysfunction whereas mini cog test had shown 15 to be involved. This difference was not statistically significant. However, assessing cognitive dysfunction in various stages of CKD did not show a statistically significant increase with the progression of CKD. In the higher stages of CKD, the assessment was confounded by the presence of encephalopathy in whom it could not be assessed. Urinary incontinence was present in 13% of the population. 40% of the study population was non ambulant requiring support for carrying out activities. This non ambulant state was attributed to fracture, severe degenerative joint disease, frailty, malnutrition, presence of encephalopathy, anemia, left ventricular dysfunction and cerebrovascular accident in the study population. Assessment of falls risk revealed 38% and 5% of the patients had severe and moderate risk of falls. These patients were initiated on prevention of falls algorithms advised by the clinical practice guidelines from the American geriatric society. The factors responsible for the high risk state were analyzed, addressed and referred to a specialist appropriately when required. Assessment of the population for frailty revealed 76% and 6% of the

population to be frail and pre-frail respectively. 2% of the patients had fracture neck of femur which had occurred after the onset and diagnosis of CKD and 45% of the patients had degenerative joint disease involving the knee, hip and shoulder joints. The degenerative joint disease and frailty had also contributed to the non ambulant state of the patient. 17% of the patients had underweight as diagnosed using Body mass index for Asian standards (<18.5). The increase in underweight as the stage of CKD progressed was not statistically significant. However, there was a progressive drop in BMI as the stage of CKD progressed. This progressive decrease in BMI with advancing stage of CKD could be attributed to uremic gastritis, catabolism aggravated by uremic state, inflammatory state, progressive decrease in hemoglobin, urinary protein loss, presence of comorbidities such as diabetes and advanced heart failure[105]

The frequency complications of CKD such as acute on CKD increased as the stage of CKD increased which was statistically significant. However, the increase in complications such as acute pulmonary edema, encephalopathy and other complications as the stage of CKD progressed was not statistically significant. None of the patients had clinical, ECG or Echo evidence of pericarditis emphasizing the rarity of the complication in the modern era and that it is more often observed in patients who are poorly dialyzed or poorly adherent to the schedule

rather than in those starting dialysis.

The analysis in acid base balance revealed increased incidence of acidosis as the stage of CKD progressed which was also evident from the progressive decrease in pH as the eGFR declined. The bicarbonate levels also decreased substantially as the eGFR declined. The acidosis is due to retention of hydrogen ions, organic acids and increased excretion of bicarbonate. The prevalence of hyponatremia and the levels of sodium did not vary as the CKD progressed. The prevalence of hyperkalemia progressively increased and the levels of potassium level progressively increased with increasing stages of CKD. The hyperkalemia in the study population in advanced stages could be attributed to the progressive increase in acidosis as evidenced by decreasing pH and decreasing bicarbonate, anemia for which blood was transfused, diet high in potassium, acute on chronic kidney disease, diabetes and drugs including potassium sparing diuretics prescribed for cardiovascular disease and ACE inhibitors. However, there was no difference in the prevalence of hyperkalemia and the potassium levels in the serum between diabetic and non diabetic patients. This expected observation occurs due to the added factor of hyporeninemic hypoaldosteronism. The lack of this difference could be explained by the fact that such differences will be exaggerated and obvious in the early stages of CKD 1 and 2 where the contribution of CKD in the development of hyperkalemia would be limited[107].

The mean hemoglobin concentration of 9.2 g/dl. The prevalence of anemia in the study population was almost pervasive involving 93% males and 89% females. The anemia was normocytic in 67% of the population and microcytic in 33% of the population. The mean hemoglobin concentration had progressively decreased with advancing CKD which was statistically significant.

The mean aspartate transaminase and alanine transaminase levels 24.5 and 24.1. However, the expected observation of decreased AST and ALT were not observed due to the presence of confounding factors that could cause the elevation of liver enzymes. These include diabetes mellitus and hypercholesterolemia leading to occurrence of Non alcoholic fatty liver disease which were subclinical , drugs including statins and sepsis leading on to organ dysfunction. The mean alkaline phosphatase level was 107.5

The mean of ejection fraction of the left ventricle was 53.5%. 39%, 7% and 7% had mild, moderate and severe left ventricular dysfunction. However, the difference in systolic dysfunction between groups did not reach statistical significance due to insufficient data on ejection fraction in the study population.

Further in the study population 2 patients were already on hemodialysis initiates at a private center with private funding. The vascular access had been established in the IJV for both the patients. 2

patients had been started on PD and later converted to HD, 20% of the patients were already on or were started on peritoneal dialysis. The funding for the dialysis were institutional.

The distribution of the etiology and contributory factors include 61% patients with diabetes mellitus, 75% patients with hypertension, 19% patients with chronic glomerulonephritis, 8% patients with chronic interstitial nephritis, 3 patients with items nephropathy, 2 patients with autosomal dominant polycystic kidney disease, 1 patient with myeloma kidney and the cause could not be ascertained in 4% of the population.

Subsequent follow up of the patients had shown that 38.6% of the urban population and 39.5% could not be contacted either due to loss of communication or the death of the patient. 17.14% from the urban population and 57.6% in the rural population did not follow up for medical consultation. This difference between the rural and urban population in follow up was statistically significant. The reasons cited by the patients and / or their relatives for not following up in the consultation services were due to indirect expenses incurred by the patient due to stay and travel and social stigma of the geriatric population. However, such decrease in follow up in rural population could be further attenuated by extending nephrology consultation services across the state.

CONCLUSION

The analysis of the CKD patients has shown that Diabetes mellitus and systemic hypertension are the most common etiology and contributory factor affecting three – fourths of the study population. The various complications of the geriatric population including frailty, cognitive dysfunction and fall risk were higher in CKD patients as compared to the risk at the general population level. The complications pertaining to CKD such as malnutrition, hemoglobin, acidosis, hyperkalemia and acute on CKD had shown progressive worsening and increased incidence with the advancing stages of CKD. However, the prevalence of hyponatremia, encephalopathy did not show progressive increase as the stage of CKD progressed. This poor correlation with the stages of CKD in hyponatremia and encephalopathy could be due to limited sample size.

The rural patients were lost to follow up as compared to urban population due to increased indirect expenses and social stigma associated with the Geriatric population. Hence forth extending nephrology consultation services to the rural population is essential and further studies focusing on the factors contributing to the follow up attenuation and factors that could be done to improve the follow up.

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ANNEXURES

ABBREVIATIONS USED IN TEXT

CKD - Chronic kidney disease

AER - Albumin excretion rate

NKF - National Kidney Foundation

KDOQI - kidney Disease Outcome Qualitative Initiative

KDIGO - Kidney Disease: Improving Global Outcomes

GFR - Glomerular Filtration Rate

eGFR - Estimated Glomerular Filtration Rate

MDRD - Modification of Diet in Renal Disease

CKD- EPI - Chronic Kidney Disease Epidemiology Collaboration

ADPKD - Autosomal dominant polycystic kidney disease

ACE - Angiotensin Converting Enzyme

ARB - Angiotensin Receptor Blocker

PCR - Protein Creatinine Ratio

RRT - Renal Replacement Therpay

AVF - Arterio Venous Fistula

ESRD – End Stage Renal Disease

DM – Diabetes Mellitus

AGEP – Advanced Glycation End Product

MPGN – Membranoproliferative Glomerulonephritis.

MMSE – Mini Mental State Examination

ESA – Erythropoietic Stimulating Agents

PROFORMA

STUDY OF CKD IN GERIATRIC POPULATION – ETIOLOGY, CLINICAL PROFILE AND OUTCOME

NAME OF THE PATIENT :

AGE / SEX :

IP/OP NUMBER :

OCCUPATION :

ADDRESS :

CONTACT NUMBER :

CARE GIVER :

COMPLAINTS :

PAST HISTORY : Diabetes mellitus:

Systemic hypertension:

H/O acute kidney injury:

Others:

TREATMENT HISTORY: Alternative medicine intake :

NSAID use :

DRUG ALLERGY :

PERSONAL HISTORY : Smoking : Alcohol:

Other substance abuse:

GENERAL EXAMINATION :

BMI (Quetlet's index) :

Weight : Height :

VITALS :

Blood Pressure: Pulse Rate:

Respiratory Rate: Temperature:

Ambulant : Yes / No

SYSTEMIC EXAMINATION

CARDIOVASCULAR SYSTEM:

RESPIRATORY SYSTEM :

ABDOMEN :

CENTRAL NERVOUS SYSTEM: Dementia & cognitive assessment - MMSE:

Mini cog test:

Eye & Fundus Examination:

Ear & Hearing Assessment:

Presence of severe joint disease (if present):

Duration of CKD :

Presence of :

Fraility -(Assessed by Cardiovascular health study index)

Falls & falls risk assessment -

Urinary incontinence -

INVESTIGATIONS:

Hemogram :

LFT:

Urinalysis :

Fasting Lipid Profile

RENAL FUNCTION TEST : Creatinine -

Blood urea nitrogen -

eGFR:

Cockroft Gault - MDRD - CKD EPI -

Stage of CKD:

Cockroft Gault - MDRD - CKD EPI -

Ultrasound Abdomen:

Complications at presentation : Acute on chronic kidney disease -

Acute pulmonary edema -

Acidosis -

Hyperkalemia -

Encephalopathy -

Pericarditis

Arterial blood gas: pH -

pCO2 -

Bicarbonate -

Anion Gap -

Serum electrolytes: Serum Sodium -

Serum Potassium-

Ejection Fraction :

ECG :

Chest Xray :

Viral Markers: HbsAg - antiHCV - HIV -

Special investigation (if any of the folllowing done)

CT / MRI Abdomen -

Renal Biopsy -

Others (if any done) -

Renal Replacement Therapy (RRT) : Yes / No If yes,

(i) Mode of RRT : Hemodialysis / Peritoneal dialysis / both

(ii) Centre for RRT :

(iii) Frequency of RRT :

(iv) Funding : Institutional / Insurance / Private

(v) Vascular access if patient is on hemodialysis :

ETIOLOGY : Diabetes Mellitus - Hypertension -

Chronic Glomerulonephritis -

Chronic Interstitial Nephritis -

Myeloma -

Obstructive Uropathy-

Ischemic nephropathy -

Others (mention the cause) Unknown –

MINI COG TEST

Pt. Name:_____ DOB:_____
Date:_____

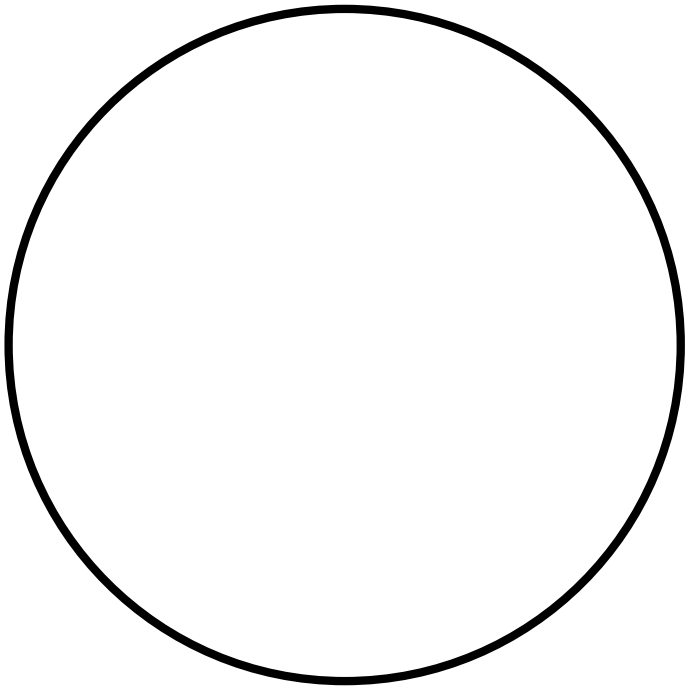
Instructions

11. Instruct the patient to listen carefully and repeat the following

2. Administer the Clock Drawing Test. Inside the circle draw the hours of a clock. Place the hands of the clock to represent the time “10 minutes past 11”

3. Ask the patient to repeat the three words given previously

_____ _____ _____



Score in Word Recall :

Score in CDT :

Interpretation :

The Mini-Mental State Exam

Patient_____ Examiner _____ Date_____

Maximum	Score	
		Orientation
5	( )	What is the (year) (season) (date) (day) (month)?
5	( )	Where are we (state) (country) (town) (hospital) (floor)?
		Registration
3	( )	Name 3 objects: 1 second to say each. Then ask the patient all 3 after you have said them. Give 1 point for each correct answer. Then repeat them until he/she learns all 3. Count trials and record. Trials _____
		Attention and Calculation
5	( )	Serial 7's. 1 point for each correct answer. Stop after 5 answers. Alternatively spell "world" backward.
		Recall
3	( )	Ask for the 3 objects repeated above. Give 1 point for each correct answer.
		Language
2	( )	Name a pencil and watch.
1	( )	Repeat the following "No ifs, ands, or buts"
3	( )	Follow a 3-stage command: "Take a paper in your hand, fold it in half, and put it on the floor."
1	( )	Read and obey the following: CLOSE YOUR EYES
1	( )	Write a sentence.
1	( )	Copy the design shown.

_____ Total Score

ASSESS level of consciousness along a continuum _____

Alert Drowsy Stupor Coma

"MINI-MENTAL STATE." A PRACTICAL METHOD FOR GRADING THE COGNITIVE STATE OF PATIENTS FOR THE CLINICIAN. *Journal of Psychiatric Research*, 12(3): 189-198, 1975. Used by permission.

Write a sentence :

Copy the design :

FRAILTY INDEX:

OSTEOPOROTIC FRACTURE INDEX:

12. Weight loss of 5 percent in last year

13. Inability to rise from a chair five times without use of arms, or

14. A "no" response to the question "Do you feel full of energy?"

INTERPRETATION

2 or more - Frailty

1 - Pre Frailty

0 - No frailty

FALLS RISK

RISK FACTOR	LEVEL		RISK SCORE
RECENT FALLS *(To score this, complete history of falls, overleaf)*	none in last 12 months.....		2
	one or more between 3 and 12 months ago.....		4
	one or more in last 3 months.....		6
	one or more in last 3 months whilst inpatient / resident....		8
MEDICATIONS *(Sedatives, Anti-Depressants Anti-Parkinson's, Diuretics Anti-hypertensives, hypnotics)*	not taking any of these.....		1
	taking one		2
	taking two		3
	taking more than two.....		4
PSYCHOLOGICAL *(Anxiety, Depression ↓Cooperation, ↓Insight or ↓Judgement **esp. re mobility**)*	does not appear to have any of these.....		1
	appears mildly affected by one or more.....		2
	appears moderately affected by one or more.....		3
	appears severely affected by one or more.....		4
COGNITIVE STATUS *(AMTS: Hodkinson Abbreviated Mental Test Score)*	AMTS 9 or 10 / 10	**OR** intact.....	1
	AMTS 7-8	mildly impaired.....	2
	AMTS 5-6	mod impaired.....	3
	AMTS 4 or less	severely impaired	4
(Low Risk: 5-11 Medium: Risk: 12-15 High Risk: 16-20)		**RISK SCORE**	/20

Automatic High Risk Status: *(if ticked then circle* **HIGH** *risk below)*
☐ Recent change in functional status and / or medications affecting safe mobility (or anticipated)
☐ Dizziness / postural hypotension

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To

Dr.Guhan.R.
Post Graduate in MD(General Medicine)
Madras Medical College
Chennai 600 003

Dear Dr.Guhan.R.

The Institutional Ethics Committee has considered your request and approved your study titled " **STUDY OF CHRONIC KIDNEY DISEASE IN GERIATRIC POPULATION – ETIOLOGY, CLINICAL PROFILE AND OUTCOME** " NO.28042015.

The following members of Ethics Committee were present in the meeting hold on 07.04.2015 conducted at Madras Medical College, Chennai 3

1. Prof.C.Rajendran, MD	:Chairperson
2. Prof.R.Vimala,MD.,Dean,MMC,Ch-3	: Deputy Chairperson
3. Prof.B.Kalaiselvi,MD.,Vice Principal,MMC,Ch-3	: Member Secretary
4. Prof.B.Vasanthi,MD.,Prof.of Pharmacology,MMC	: Member
5. Prof.Raghumani,MS.,Prof.of Surgery,MMC	:Member
6. Prof.S.Baby Vasumathi, Director, Inst. of O&G,MMC	: Member
7. Prof.K.Ramadevi,MD., Director ,Inst.of Bio-Chem.MMC	: Member
8. Prof.Saraswathy,MD.,Director,Pathology, MMC	: Member
9.Prof.K.Srinivasagalu,MD.,Director, I.I.M , MMC	: Member
10.Thiru S.Rameshkumar, B.Com., MBA.	: Lay Person
11.Thiru S.Govindasamy, BA., BL.,	: Lawyer
12.Tmt.Arnold Saulina, MA., MSW.,	: Social Scientist

We approve the proposal to be conducted in its presented form.

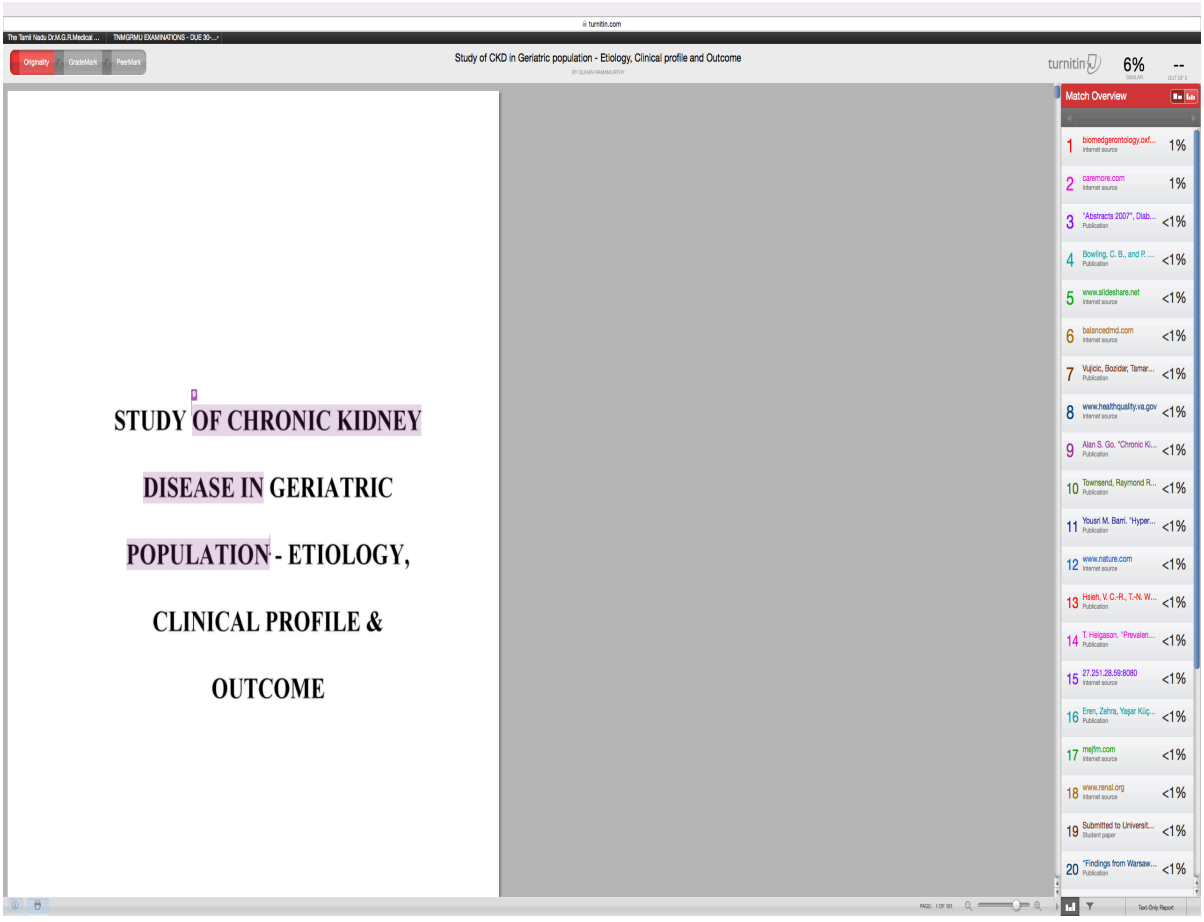
The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

Sys 2

TURNITIN – PLAGIARISM – SCREENSHOT



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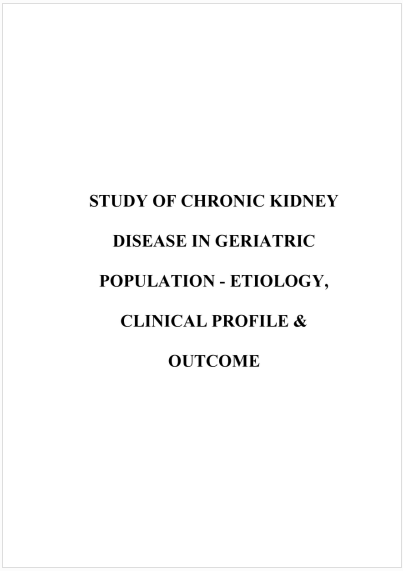

STUDY OF CHRONIC KIDNEY DISEASE IN GERIATRIC POPULATION - ETIOLOGY, CLINICAL PROFILE & OUTCOME

INFORMATION SHEET

We are conducting a study on "STUDY OF CHRONIC KIDNEY DISEASE IN GERIATRIC POPULATION - ETIOLOGY, CLINICAL PROFILE AND OUTCOME" among patients attending Rajiv Gandhi Government General Hospital, Chennai

The purpose of this study is to assess "Etiology, clinical profile and outcome in patients with chronic kidney disease in geriatric population"

We are selecting certain cases and if you are found eligible, we may be using clinical profile, lab test reports and radiological reports for study purposes which does not affect your final report or management.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator Signature of Participant / Guardian

Date :
Place :

ஆராய்ச்சி தகவல் தாள்

சென்னை இராஜிவ் காந்தி அரசு பொது மருத்துவமனையில் பற்றிய ஒரு ஆராய்ச்சி நடைபெற்று வருகிறது.

முதியோர் மக்கள் தொகையில் உள்ள நாள்பட்ட சிறுநீரக நோய்க்கான தொகுப்பே இந்த ஆராய்ச்சியின் நோக்கமாகும்.

நீங்கள் இந்த ஆய்விற்கு தகுதியானவர்களாக இருக்கும் பட்சத்தில் தங்களின் மருத்துவ சுயவிவரத்தையும், ஆய்வக பரிசோதனை அறிக்கையும் மற்றும் கதிரியக்க அறிக்கையும் ஆய்வு நோக்கங்களுக்காக பயன்படுத்தபடும். இது உங்களுடைய இறுதி அறிக்கை அல்லது மேலாண்மையை பாதிக்காது

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். முடிவுகளை அல்லது கருத்துக்களை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியிலிருத்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த சிறப்புப் பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சியின் முடிவில் தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

தேதி:

PATIENT CONSENT FORM

Study Detail	:	STUDY OF CHRONIC KIDNEY DISEASE IN GERIATRIC POPULATION - ETIOLOGY, CLINICAL PROFILE AND OUTCOME
Study Centre	:	Rajiv Gandhi Government General Hospital, Chennai.
Patient's Name	:	
Patient's Age	:	
Identification Number	:	

Patient may check (☑) these boxes

The details of the study have been provided to me in writing and explained to me in my own language ❑

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ❑

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ❑

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ❑

I hereby consent to participate in this study. ❑

I hereby give permission to undergo complete clinical examination , biochemical and radiological tests ❑

Signature of Investigator
Study Investigator's Name:
DR. GUHAN R

Signature/thumb impression
Patient's Name and Address:

ஆராய்ச்சி ஒப்புதல் கடிதம்

ஆராய்ச்சி தலைப்பு: முதியோர் மக்கள் தொகையில் உள்ள நாள்பட்ட சிறுநீரக நோய்க்கான தொகுப்பு.

ஆய்வு நிலையம் : பொது நல மருத்துவத்துறை
சென்னை மருத்துவக் கல்லூரி
சென்னை - 600003

பெயர்: தேதி:

வயது: உள்நோயாளி எண்:

பால்: ஆராய்ச்சி சேர்க்கை எண்:

ஆய்வு விவரங்களை எனது சொந்த மொழியில் எனக்கு விளக்கினார். எனக்கு சந்தேகம் கேட்க ஒரு வாய்ப்பும், அதற்கு தகுந்த பதில்களும் வழங்கப்பட்டது. ❑

நான் இந்த ஆய்வில் தன்னிச்சையாகதான் பங்ககேற்கிறேன். எந்த காரணத்திலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன். ❑

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்பதற்க்கு என் அனுமதி தேவை இல்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன் ❑

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்கள்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக்கொள்ளவும் அதை பிரசுரிக்கவும் நான் முழு மனதுடன் ஒப்புக்கொள்கிறேன் ❑

நான் ஆய்வில் பங்கேற்க ஒப்புக்கொள்கிறேன். நான் மருத்துவரிடம் உண்மையாக இருப்பேன் என உறுதியளிகிறேன் ❑

ஆய்வாளரின் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

ஆய்வாளரின் பெயர்: பங்கேற்பாளர் பெயர் முகவரி

டாக்டர். இரா. குகன்

MASTER CHART

NAME	AGE	GENDER	LOCATION	FOLLOW UP	DIABETES MELLITUS	DURATION OF DIABETES (IN YRS)	DIABETIC RETINOPATHY
Boopalan	66	M	CHENNAI	-	Y	22	NPDR
Kondaiah	65	M	OUTSIDE CHENNai	-	N	-	-
Perumal	75	M	OUTSIDE CHENNai	Y	N	-	-
Raja	68	M	CHENNAI	Y	Y	8	NPDR
Vasantha Kumari	62	F	OUTSIDE CHENNai	-	Y	10	NPDR
Ramayi	61	F	OUTSIDE CHENNai	Y	N	-	-
Angusamy	80	M	OUTSIDE CHENNai	-	N	-	-
Munusamy	60	M	CHENNAI	-	N	-	-
Parvathy	60	F	CHENNAI	Y	Y	6	NPDR
Selvi	64	F	OUTSIDE CHENNai	Y	N	-	-
Abdul Jaffar	62	M	CHENNAI	Y	N	-	-
Ramaih	70	F	CHENNAI	Y	N	-	-
Shanthi	65	F	OUTSIDE CHENNai	Y	N	-	-
Savitri	60	F	OUTSIDE CHENNai	-	N	-	-
Shenbagavalli	63	F	CHENNAI	-	Y	7	NPDR
Parameshwari	65	F	CHENNAI	-	Y	10	NPDR
Jagadishwari	60	F	OUTSIDE CHENNai	-	Y	7	-
Selvam	60	M	CHENNAI	-	Y	0.5	-
Elizabeth	70	F	CHENNAI	Y	Y	1	-
Subhamma	60	F	CHENNAI	N	N	-	-
Poongothai	67	F	OUTSIDE CHENNai	-	Y	15	NPDR
Angaiyan	62	M	CHENNAI	Y	N	-	-
Kuppammal	65	F	CHENNAI	-	Y	7	NPDR
Panjammal	70	F	OUTSIDE CHENNai	Y	Y	9	NPDR
Ranganathan	63	M	OUTSIDE CHENNai	N	Y	5	NPDR
Vasantha	65	F	CHENNAI	-	N	-	-
Sakunthala	70	F	CHENNAI	Y	N	-	-
Periasamy	70	M	CHENNAI	Y	N	-	-
Pandi	60	M	CHENNAI	N	N	-	-
Ramu	69	M	OUTSIDE CHENNai	Y	Y	4	NPDR
Chinnaponnu	70	F	OUTSIDE CHENNai	-	N	-	-
Vazeer Baasha	78	M	CHENNAI	-	Y	10	-
Prakash	60	M	CHENNAI	Y	Y	5	NPDR
Vasantha	60	F	OUTSIDE CHENNai	N	Y	6	NPDR
Periyasamy	78	M	OUTSIDE CHENNai	Y	N	-	-
Vanitha	79	F	CHENNAI	N	Y	20	-
Krishnakumari	65	F	CHENNAI	-	Y	20	NPDR
Murugesan	60	M	OUTSIDE CHENNai	Y	N	-	-
Petchiyammal	62	F	OUTSIDE CHENNai	N	Y	10	NPDR
Gopal	60	M	OUTSIDE CHENNai	N	N	-	-
Sunthari	62	F	CHENNAI	-	Y	10	NPDR
Saraswathi	65	F	CHENNAI	Y	N	-	-
Lakshmi	69	F	CHENNAI	Y	Y	5	NPDR
Mohammed Hussaun	62	M	OUTSIDE CHENNai	Y	Y	12	NPDR
Chinnayyan	63	M	CHENNAI	-	Y	4	-
Sambasivam	70	M	CHENNAI	Y	Y	6	-
Alankaram	67	F	CHENNAI	-	N	-	-
Egambaram	77	M	CHENNAI	Y	Y	1	-
Dharman	70	M	OUTSIDE CHENNai	-	Y	5	-
Parthasarathy	62	M	OUTSIDE CHENNai	Y	Y	20	NPDR

MASTER CHART

NAME	AGE	GENDER	LOCATION	FOLLOW UP	DIABETES MELLITUS	DURATION OF DIABETES (IN YRS)	DIABETIC RETINOPATHY
Dhanapal	62	M	CHENNAI	-	Y	20	NPDR
Lakshmi	60	F	CHENNAI	Y	N	-	-
Ellaiyammal	65	F	OUTSIDE CHENNai	-	Y	3	-
Ramamoorthy	60	M	CHENNAI	Y	Y	4	-
Kuppusamy	62	M	CHENNAI	Y	N	-	-
Dhanalakshmi	68	F	OUTSIDE CHENNai	N	Y	10	NPDR
Poongavanam	67	F	CHENNAI	-	Y	0.5	NPDR
Fathima	70	F	CHENNAI	Y	N	-	-
Babu	60	M	OUTSIDE CHENNai	N	Y	5	NPDR
Anbumani	61	F	OUTSIDE CHENNai	N	Y	25	NPDR
Indrani	65	F	CHENNAI	Y	Y	2	NPDR
Sampath	61	M	OUTSIDE CHENNai	-	N	-	-
Krishnaveni	60	F	CHENNAI	Y	Y	11	NPDR
Ravi	64	M	OUTSIDE CHENNai	N	N	-	-
Davlath Ram	78	M	CHENNAI	Y	Y	8	-
Ravi	60	M	CHENNAI	N	Y	7	NPDR
Indirani	74	F	CHENNAI	-	Y	1	-
Kamala	60	F	CHENNAI	N	Y	17	NPDR
Duraimani	66	M	CHENNAI	-	Y	10	NPDR
Saida	61	F	CHENNAI	Y	Y	5	-
Valiyammal	65	F	CHENNAI	Y	Y	20	-
Goundhan	62	M	OUTSIDE CHENNai	-	Y	10	NPDR
Kalathari	65	F	CHENNAI	N	Y	7	NPDR
Ravi	61	M	OUTSIDE CHENNai	N	Y	5	-
Rajammal	60	F	CHENNAI	Y	Y	5	-
Gangaiammal	65	F	CHENNAI	-	Y	6	NPDR
Chellamal	70	F	CHENNAI	Y	Y	10	-
Gajalakshmi	60	F	CHENNAI	Y	N	-	-
Valliyammal	62	F	OUTSIDE CHENNai	-	N	-	-
Gangammal	60	F	CHENNAI	-	Y	30	-
Janardhanan	61	M	CHENNAI	-	Y	18	NPDR
Velan	70	M	OUTSIDE CHENNai	Y	Y	10	-
Ezhumalai	67	M	OUTSIDE CHENNai	N	N	-	-
Murugesan	74	M	CHENNAI	Y	N	-	-
Jayaraj	60	M	OUTSIDE CHENNai	-	Y	15	-
Mani	60	M	OUTSIDE CHENNai	N	Y	12	NPDR
Venkatammal	64	F	CHENNAI	-	N	-	NPDR
?Arunmiyam	65	M	OUTSIDE CHENNai	N	Y	5	-
Rani	67	F	CHENNAI	-	Y	20	PDR
Rajendran	66	M	CHENNAI	Y	N	-	-
Ponnamal	66	F	OUTSIDE CHENNai	N	Y	8	NPDR
Mary	61	F	CHENNAI	Y	N	-	-
Muniyammal	75	F	OUTSIDE CHENNai	-	N	-	-
Jyothi	60	F	OUTSIDE CHENNai	-	N	-	-
kuppusamy	66	M	OUTSIDE CHENNai	-	N	-	-
Mani	68	M	OUTSIDE CHENNai	N	Y	8	B/L NPDR
Munimmal	64	F	OUTSIDE CHENNai	-	N	-	-
Radha	65	F	CHENNAI	-	Y	6	-
Raniammal	62	F	OUTSIDE CHENNai	N	N	-	-
Nagarajan	65	M	CHENNAI	Y	Y	5	NPDR

MASTER CHART

NAME	SYSTEMIC HTN	DURATION OF SYSTEMIC HTN (IN YRS)	PRESENCE OF HYPERTENSIVE RETINOPATHY	SMOKER	ALCOHOLIC	DURATION OF CKD (IN YRS)	CREATININE (MG/DL)
Boopalan	N			N	N	2	3
Kondaiah	N			Y	N	1	1.4
Perumal	Y	4.5		N	N	1	3
Raja	Y	6	Y	Y	Y	1	3.2
Vasantha Kumari	Y	5	Y	N	N	3	1.9
Ramayi	Y	1		N	N	1	22.9
Angusamy	N			Y	Y	6	8.7
Munusamy	Y	2		N	Y	2	9.4
Parvathy	Y	6		N	N	1	1.8
Selvi	Y	4	Y	N	N	1	8.3
Abdul Jaffar	Y	2		Y	Y	0.5	7.6
Ramaih	N			N	N	0.25	22.8
Shanthi	N			N	N	0.5	6.1
Savitri	Y	0.5	Y	N	N	0.75	7.7
Shenbagavalli	Y	7		N	N	1	6.9
Parameshwari	Y	2		N	N	1	5.9
Jagadishwari	Y	1		N	N	0.5	9.5
Selvam	N			N	Y	9	2.4
Elizabeth	Y	1		N	N	1	3.2
Subhamma	Y	1		N	N	1	5.1
Poongothai	Y	7	Y	N	N	5	6.4
Angaiyan	N			Y	Y	1	3
Kuppammal	Y	5		N	N	1	1.9
Panjammal	Y	0.5		N	N	1	3.4
Ranganathan	Y	5		Y	Y	3	10.4
Vasantha	Y	8		N	N	8	12.1
Sakunthala	Y	10	Y	N	N	1	2.6
Periasamy	Y	5		N	Y	1	1.7
Pandi	Y	20	Y	N	Y	1	1.5
Ramu	Y	1		N	N	0.5	12.7
Chinnaponnu	Y	1		N	N	0.25	3.7
Vazeer Baasha	Y	10		N	N	0.5	1.6
Prakash	Y	3		N	N	3	9.7
Vasantha	Y	6		N	N	1	2.2
Periyasamy	N			N	N	0.5	5.3
Vanitha	N			N	N	0.5	1.4
Krishnakumari	Y	10		N	N	5	3.4
Murugesan	Y	5	Y	N	Y	1	3
Petchiyammal	Y	4		N	N	2	2.3
Gopal	Y	1		N	N	0.5	5.1
Sunthari	N			N	N	1	4.1
Saraswathi	N			N	N	2	2.1
Lakshmi	N			N	N	1	1.3
Mohammed Hussaun	Y	0.75		Y	N	0.5	4.5
Chinnayyan	Y	2		N	Y	1	4.9
Sambasivam	Y	3		N	N	1	2.3
Alankaram	N			N	N	0.5	2.6
Egambaram	N			Y	N	1	2.4
Dharman	N			N	N	1	1.4
Parthasarathy	Y	0.75		N	Y	1.5	7.7

MASTER CHART

NAME	SYSTEMIC HTN	DURATION OF SYSTEMIC HTN (IN YRS)	PRESENCE OF HYPERTENSIVE RETINOPATHY	SMOKER	ALCOHOLIC	DURATION OF CKD (IN YRS)	CREATININE (MG/DL)
Dhanapal	Y	0.25		Y	Y	0.75	19
Lakshmi	Y	14		N	N	1	1.4
Ellaiyammal	Y	3		N	N	1	4.2
Ramamoorthy	Y	4		Y	Y	1	9.7
Kuppusamy	N			Y	Y	3	5
Dhanalakshmi	Y	10		N	N	2	1.9
Poongavanam	N			N	N	0.5	2.8
Fathima	N			N	N	0.5	4.6
Babu	Y	1		Y	N	1	1.7
Anbumani	Y	20	Y	N	N	1	1.6
Indrani	Y	0.5		N	N	0.5	1.7
Sampath	Y	14	Y	Y	N	14	8.2
Krishnaveni	Y	3		N	N	0.5	1.4
Ravi	N			N	Y	0.5	3.6
Davlath Ram	Y	8		N	N	2	3.5
Ravi	Y	4		N	Y	2	1.7
Indirani	Y	1		N	N	1	2.8
Kamala	Y	3		N	N	0.25	2.2
Duraimani	Y	5		Y	Y	2	6.6
Saida	Y	5		N	N	0.5	1.6
Valiyammal	N			N	N	1	1.9
Goundhan	Y	8		N	Y	3	4.3
Kalathari	N			N	N	0.25	1.5
Ravi	Y	5	Y	Y	Y	0.25	3.6
Rajammal	Y	3		Y	Y	0.25	2.4
Gangaiammal	Y	2		N	N	1	3.6
Chellamal	Y	10		N	N	0.25	7.1
Gajalakshmi	Y	5		N	N	0.25	1.5
Valliyammal	N			N	N	0.75	2.5
Gangammal	Y	10		N	N	1	4.9
Janardhanan	Y	0.75		N	N	0.75	7.9
Velan	Y	10		N	N	2	8.5
Ezhumalai	Y	3	Y	N	N	1	1.4
Murugesan	Y	14	Y	N	Y	1	2.7
Jayaraj	Y	15	Y	Y	Y	2	4.4
Mani	Y	8	Y	Y	Y	0.75	1.4
Venkatammal	Y	0.5		N	N	0.5	2.9
?Arunmiyam	Y	5		Y	Y	0.5	3.2
Rani	Y	20	Y	N	N	0.5	2.7
Rajendran	N			N	N	0.25	3.2
Ponnamal	Y	8		N	N	1	1.7
Mary	Y	10	Y	N	N	1	2.2
Muniyammal	Y	5		N	N	2	4.1
Jyothi	N			N	N	1.5	3.3
kuppusamy	Y	3		Y	Y	1.5	3.4
Mani	Y	3		Y	N	2	2.9
Munimmal	Y	3		N	N	1	2.6
Radha	Y	2		N	N	2	3.4
Raniammal				N	N	1.5	1.9
Nagarajan	Y	3		N	N	1	1.8

MASTER CHART

NAME	STAGING OF CKD by CKD - EPI	Weight	Height (in cm)	Height (in m)	BMI	Underweight	AMBULANT
Boopalan	4	68	170	1.7	23.5294117647059	N	N
Kondaiah	3a	62	170	1.7	21.4532871972318	N	Y
Perumal	4	62	168	1.68	21.9671201814059	N	N
Raja	4	80	168	1.68	28.3446712018141	N	N
Vasantha Kumari	4	32	146	1.46	15.0121974103959	Y	Y
Ramayi	5	52	152	1.52	22.5069252077562	N	Y
Angusamy	5	42	166	1.66	15.2416896501669	Y	Y
Munusamy	5	45	164	1.64	16.7311124330756	Y	N
Parvathy	3b	41	155	1.55	17.0655567117586	Y	Y
Selvi	5	45	162	1.62	17.1467764060357	Y	N
Abdul Jaffar	5	49	168	1.68	17.3611111111111	Y	Y
Ramaih	5	46	162	1.62	17.5278158817253	Y	N
Shanthi	5	47	163	1.63	17.6897888516692	Y	N
Savitri	5	50	168	1.68	17.7154195011338	Y	N
Shenbagavalli	5	53	172	1.72	17.9150892374256	Y	Y
Parameshwari	5	48	162	1.62	18.2898948331047	N	N
Jagadishwari	5	58	178	1.78	18.3057694735513	N	Y
Selvam	4	45	156	1.56	18.491124260355	N	Y
Elizabeth	5	51	166	1.66	18.5077660037741	N	Y
Subhamma	5	44	153	1.53	18.796189499765	N	Y
Poongothai	5	56	172	1.72	18.9291508923743	N	N
Angaiyan	4	52	164	1.64	19.3337299226651	N	N
Kuppammal	4	45	151	1.51	19.7359764922591	N	Y
Panjammal	5	52	162	1.62	19.8140527358634	N	Y
Ranganathan	5	58.4	168	1.68	20.6916099773243	N	N
Vasantha	5	48	152	1.52	20.7756232686981	N	N
Sakunthala	4	55	162	1.62	20.9571711629325	N	Y
Periasamy	3b	55	162	1.62	20.9571711629325	N	Y
Pandi	3a	55	168	1.68	19.4869614512472	N	Y
Ramu	5	58	166	1.66	21.0480476121353	N	Y
Chinnaponnu	5	58	166	1.66	21.0480476121353	N	N
Vazeer Baasha	3b	60	168	1.68	21.2585034013605	N	N
Prakash	5	50	153	1.53	21.359306249733	N	Y
Vasantha	4	58	164	1.64	21.5645449137418	N	N
Periyasamy	5	61	168	1.68	21.6128117913832	N	Y
Vanitha	3b	52	155	1.55	21.6441207075963	N	N
Krishnakumari	5	60	166	1.66	21.7738423573813	N	Y
Murugesan	4	56	160	1.6	21.875	N	Y
Petchiyammal	4	52	154	1.54	21.9261258222297	N	Y
Gopal	5	65	172	1.72	21.9713358572201	N	N
Sunthari	5	55	158	1.58	22.0317256849864	N	N
Saraswathi	4	51	152	1.52	22.0740997229917	N	Y
Lakshmi	3b	58	162	1.62	22.1002895900015	N	N
Mohammed Hussaun	5	64	170	1.7	22.1453287197232	N	N
Chinnayyan	5	70	176	1.76	22.5981404958678	N	N
Sambasivam	4	65	168	1.68	23.0300453514739	N	Y
Alankaram	5	62	164	1.64	23.051754907793	N	N
Egambaram	4	62	164	1.64	23.051754907793	N	N
Dharman	3a	72	175	1.75	23.5102040816327	N	N
Parthasarathy	5	68	170	1.7	23.5294117647059	N	N

MASTER CHART

NAME	STAGING OF CKD by CKD - EPI	Weight	Height (in cm)	Height (in m)	BMI	Underweight	AMBULANT
Dhanapal	5	72	174	1.74	23.7812128418549	N	Y
Lakshmi	3b	72	174	1.74	23.7812128418549	N	N
Ellaiyammal	5	58	156	1.56	23.8330046022354	N	N
Ramamoorthy	5	76	178	1.78	23.9868703446535	N	Y
Kuppusamy	5	78	180	1.8	24.0740740740741	N	Y
Dhanalakshmi	4	58	168	1.68	20.5498866213152	N	Y
Poongavanam	5	62	167	1.67	22.2309871275413	N	N
Fathima	5	62	160	1.6	24.21875	N	Y
Babu	3b	70	170	1.7	24.2214532871972	N	Y
Anbumani	3b	54	148	1.48	24.6530314097882	N	Y
Indrani	3b	65	162	1.62	24.7675659198293	N	Y
Sampath	5	65	162	1.62	24.7675659198293	N	Y
Krishnaveni	3b	62	158	1.58	24.8357634994392	N	Y
Ravi	4	63	174	1.74	20.8085612366231	N	N
Davlath Ram	4	65	176	1.76	20.9839876033058	N	N
Ravi	3b	68	172	1.72	22.9853975121687	N	Y
Indirani	4	75	172	1.72	25.3515413737155	N	y
Kamala	4	63	168	1.68	22.3214285714286	N	Y
Duraimani	5	75	170	1.7	25.9515570934256	N	N
Saida	3b	68	170	1.7	23.5294117647059	N	Y
Valiyammal	4	56	155	1.55	23.309053069719	N	Y
Goundhan	5	72	170	1.7	24.9134948096886	N	N
Kalathari	3b	65	155	1.55	27.0551508844953	N	Y
Ravi	4	63	165	1.65	23.1404958677686	N	Y
Rajammal	4	62	168	1.68	21.9671201814059	N	Y
Gangaiammal	5	68	160	1.6	26.5625	N	Y
Chellamal	5	82	166	1.66	29.7575845550878	N	Y
Gajalakshmi	3b	76	172	1.72	25.6895619253651	N	Y
Valliyammal	4	47	153	1.53	20.077747874749	N	N
Gangammal	5	58	168	1.68	20.5498866213152	N	Y
Janardhanan	5	56	174	1.74	18.4964988769983	N	N
Velan	5	53	172	1.72	17.9150892374256	Y	?
Ezhumalai	3a	48	165	1.65	17.6308539944904	Y	Y
Murugesan	4	58	163	1.63	21.8299521999323	N	Y
Jayaraj	5	78	168	1.68	27.6360544217687	N	Y
Mani	3a	72	172	1.72	24.3374797187669	N	Y
Venkatammal	4	58	166	1.66	21.0480476121353	N	N
?Arunmiyam	4	70	166	1.66	25.4028160836116	N	Y
Rani	4	48	168	1.68	17.0068027210884	Y	Y
Rajendran	4	62	170	1.7	21.4532871972318	N	N
Ponnamal	3b	65	168	1.68	23.0300453514739	N	Y
Mary	4	40	158	1.58	16.0230732254446	Y	
Muniyammal	5	38	156	1.56	15.6147271531887	Y	Y
Jyothi	5	72	168	1.68	25.5102040816327	N	N
kuppusamy	4	48	172	1.72	16.2249864791779	Y	N
Mani	4	55	168	1.68	19.4869614512472	N	y
Munimmal	4	52	162	1.62	19.8140527358634	N	Y
Radha	5	48	165	1.65	17.6308539944904	Y	N
Raniammal	4	58	166	1.66	21.0480476121353	N	Y
Nagarajan	3b	72	172	1.72	24.3374797187669	N	Y

MASTER CHART

NAME	COGNITIVE DYSFUNCTION ASSESSED BY MMSE	COGNITIVE DYSFUNCTION ASSESSED BY MINI COG TEST	B/L SNHL	PRESENCE OF JOINT DISEASE	PRESENCE OF FRAILTY	FALLS RISK	URINARY INCONTINENCE
Boopalan	Y	Y	Y	# R neck of femur 1yr ago	YES	HIGH	-
Kondaiah	N	N	Y	B/L OA	YES	LOW	-
Perumal	NA	NA	Y		YES	HIGH	-
Raja	NA	NA	-		YES	HIGH	-
Vasantha Kumari	N	N	-	B/L OA	YES	MODERATE	-
Ramayi	N	N	-			LOW	-
Angusamy	N	N	Y		YES	LOW	-
Munusamy	NA	NA	Y	B/L OA	YES	HIGH	-
Parvathy	N	N	Y	B/L OA	YES	HIGH	YES
Selvi	NA	NA	-		YES	HIGH	-
Abdul Jaffar	N	N	-		YES	LOW	-
Ramaih	Y	Y	Y	B/L OA	YES	MODERATE	-
Shanthi	NA	NA	Y	B/L OA	YES	HIGH	-
Savitri	N	N	-			LOW	-
Shenbagavalli	N	N	Y			LOW	-
Parameshwari	N	N	-			LOW	-
Jagadishwari	N	N	-		YES	LOW	-
Selvam	N	N	-	B/L OA	YES	HIGH	-
Elizabeth	N	N	-			HIGH	-
Subhamma	N	N	-	B/L OA		HIGH	-
Poongothai	NA	NA	-		YES	HIGH	-
Angaiyan	Y	Y	Y	B/L OA	YES	LOW	-
Kuppammal	Y	N	Y	B/L OA	YES	LOW	YES
Panjammal	N	N	-	B/L OA	YES	LOW	-
Ranganathan	NA	NA	-			HIGH	-
Vasantha	Y	Y	-		YES	HIGH	-
Sakunthala	N	N	Y	B/L OA	YES	LOW	YES
Periasamy	Y	Y	Y		YES	LOW	-
Pandi	Y	Y	-		YES	MEDIUM	-
Ramu	Y	Y	Y	B/L OA	YES	LOW	-
Chinnaponnu	Y	Y	Y	B/L OA	YES	LOW	YES
Vazeer Baasha	Y	Y	Y	B/L OA	YES	HIGH	YES
Prakash	N	N	-		YES	HIGH	-
Vasantha	N	N	-	B/L OA	YES	HIGH	-
Periyasamy	N	N	-		YES	MODERATE	-
Vanitha	Y	Y	-	B/L OA	YES	LOW	-
Krishnakumari	N	N	-			HIGH	-
Murugesan	N	N	-			HIGH	-
Petchiyammal	Y	Y	Y	B/L OA	YES	LOW	-
Gopal	NA	NA	-	B/L OA	YES	HIGH	-
Sunthari	NA	NA	-	B/L OA	YES	HIGH	-
Saraswathi	N	N	Y		YES	LOW	-
Lakshmi	N	N	-	B/L OA	YES	LOW	YES
Mohammed Hussaun	Y	N	-		YES	LOW	-
Chinnayyan	N	N	-			LOW	-
Sambasivam	N	N	-		YES	LOW	-
Alankaram	N	N	Y	B/L OA	YES	LOW	YES
Egambaram	NA	NA	Y	B/L OA	YES	HIGH	YES
Dharman	NA	NA	-	B/L OA	YES	HIGH	-
Parthasarathy	NA	NA	Y	B/L OA	YES	HIGH	-

MASTER CHART

NAME	COGNITIVE DYSFUNCTION ASSESSED BY MMSE	COGNITIVE DYSFUNCTION ASSESSED BY MINI COG TEST	B/L SNHL	PRESENCE OF JOINT DISEASE	PRESENCE OF FRAILTY	FALLS RISK	URINARY INCONTINENCE
Dhanapal	N	N	-		YES	LOW	-
Lakshmi	NA	NA	Y		YES	HIGH	-
Ellaiyammal	N	N	-	B/L OA	YES	LOW	-
Ramamoorthy	N	N	-		YES	LOW	-
Kuppusamy	N	N	Y		YES	LOW	-
Dhanalakshmi	N	N	-	B/L OA	YES	LOW	-
Poongavanam	NA	NA	Y	B/L OA	YES	HIGH	-
Fathima	Y	Y	-			LOW	-
Babu	N	N	-			LOW	-
Anbumani	N	N	Y	B/L OA	YES	LOW	-
Indrani	N	N	-	B/L OA	YES	LOW	-
Sampath	N	N	-			LOW	-
Krishnaveni	N	N	Y	B/L OA	YES	LOW	-
Ravi	N	N	Y		YES	HIGH	-
Davlath Ram	NA	NA	Y	B/L OA	YES	HIGH	-
Ravi	N	N	Y	B/L OA	YES	LOW	-
Indirani	N	N	-		yes	HIGH	-
Kamala	N	N	Y	B/L OA	YES	LOW	-
Duraimani	NA	NA	Y	Osteoporosis # L NOF	YES	HIGH	YES
Saida	N	N	Y	B/L OA	YES	LOW	-
Valiyammal	N	N	-			LOW	-
Goundhan	NA	NA	-	B/L OA	YES	HIGH	-
Kalathari	N	N	Y	B/L OA	YES	LOW	YES
Ravi	N	N	-		YES	LOW	YES
Rajammal	N	N	-		YES	LOW	-
Gangaiammal	N	N	-			LOW	-
Chellamal	Y	Y	-			LOW	-
Gajalakshmi	N	N	Y			LOW	-
Valliyammal	N	N	-			LOW	-
Gangammal	N	N	-			LOW	-
Janardhanan	N	N	Y	B/L OA	YES	LOW	-
Velan	NA	NA	NA		YES	HIGH	-
Ezhumalai	N	N	Y		YES	LOW	-
Murugesan	N	N	Y	B/L OA	YES	LOW	-
Jayaraj	N	N	-			LOW	-
Mani	N	N	-		YES	LOW	-
Venkatammal	N	N	-	B/L OA	YES	LOW	YES
?Arunmiyam	N	N	Y		YES	LOW	-
Rani	N	N	Y			LOW	-
Rajendran	Y	Y	-		YES	LOW	-
Ponnamal	N	N	Y	B/L OA	YES	LOW	-
Mary	N	N	-			LOW	-
Muniyammal	N	N	Y			LOW	-
Jyothi	N	N	-			LOW	-
kuppusamy	N	N	Y	B/L OA	YES	LOW	-
Mani	N	N	Y		YES	LOW	-
Munimmal	N	N	-		YES	LOW	-
Radha	Y	Y	-	B/L OA	YES	LOW	YES
Raniammal	N	N	Y	B/L OA	YES	LOW	-
Nagarajan	N	N	Y	B/L OA	YES	LOW	-

MASTER CHART

NAME	HEMOGLOBIN	TYPE OF ANEMIA (M- MICROCYTOSIS; N- NORMOCYTIC)	MEAN CORPUSCULAR VOLUME	SGOT	SGPT	ALKALINE PHOSPHATASE	CREATININE
Boopalan	10	N	80.1	13	11	331	3
Kondaiah	8.4	N	80.4	13	15	104	1.4
Perumal	11.7	N	85.9	13	11	62	3
Raja	5.3	N	87.1	36	21	21	3.2
Vasantha Kumari	8.4	M	78.3	13	14	78	1.9
Ramayi	5.7	N	85.5	12	10	156	22.9
Angusamy	7.8	M	74	18	16	45	8.7
Munusamy	6.5	N	84.7	13	11	34	9.4
Parvathy	12.9	N	82.9	17	18	72	1.8
Selvi	5.9	M	78.9	27	29	110	8.3
Abdul Jaffar	6.4	N	85.5	18	62	110	7.6
Ramaih	5.7	N	85.5	12	10		22.8
Shanthi	9.6	M	74	16	19	112	6.1
Savitri	6.8	N	90	104	54	127	7.7
Shenbagavalli	11.8	M	76	30	22	99	6.9
Parameshwari	8.7	N	93.1	13	16	88	5.9
Jagadishwari	8.1	M	75	17	14	59	9.5
Selvam	9.9	M	76.4	19	18	356	2.4
Elizabeth	10.2	N	83.3	18	22	77	3.2
Subhamma	8	N	88.6	18	17	165	5.1
Poongothai	7.4	N	83.3	20	19	116	6.4
Angaiyan	6.5	N	89.8	17	18	140	3
Kuppammal	10.3	M	77.6	31	43	43	1.9
Panjammal	5.5	M	57	20	14	122	3.4
Ranganathan	6.8	M	68	16	18	98	10.4
Vasantha	5.8	M	77.8	29	35	143	12.1
Sakunthala	7.8	N	94.8	21	17	78	2.6
Periasamy	10.2	N	82	40	36	117	1.7
Pandi	14.4	N	97	23	26	49	1.5
Ramu	6.3	N	91.3	21	24	57	12.7
Chinnaponnu	11	N	85.4	27	29	153	3.7
Vazeer Baasha	12	N	84.6	23	21	151	1.6
Prakash	5.8	N	82	8	7	470	9.7
Vasantha	9.7	N	84.2	13	11	72	2.2
Periyasamy	8.5	M	74	20	21	82	5.3
Vanitha	11.3	N	87	11	13	117	1.4
Krishnakumari	8.7	N	84.6	62	38	150	3.4
Murugesan	7.6	M	72.4	24	26	28	3
Petchiyammal	12	N	84.6	17	11	70	2.3
Gopal	9.6	N	91.8	18	30	106	5.1
Sunthari	8.1	N	82.7	12	18	125	4.1
Saraswathi	8	M	73.4	14	14	145	2.1
Lakshmi	9.9	N	83.5	25	30	71	1.3
Mohammed Hussaun	13.7	N	90	11	20	177	4.5
Chinnayyan	7.5	M	68	29	13	62	4.9
Sambasivam	10.2	N	88.8	16	24	66	2.3
Alankaram	8.2	M	55.6	12	31	90	2.6
Egambaram	12.5	N	84.3	81	48	60	2.4
Dharman	11.2	N	86.4	14	13	48	1.4
Parthasarathy	9.6	N	89.8	29	24	174	7.7

MASTER CHART

NAME	HEMOGLOBIN	TYPE OF ANEMIA (M- MICROCYTOSIS; N- NORMOCYTIC)	MEAN CORPUSCULAR VOLUME	SGOT	SGPT	ALKALINE PHOSPHATASE	CREATININE
Dhanapal	7.2	M	78	14	12		19
Lakshmi	12.1	N	82.7	20	57	50	1.4
Ellaiyammal	6.8	M	79.6	26	23	56	4.2
Ramamoorthy	6.6	M	64.2	12	16	16	9.7
Kuppusamy	7.9	N	94.2	31	33	67	5
Dhanalakshmi	9.9	N	82.8	18	19	79	1.9
Poongavanam	8	M	78	30	45	75	2.8
Fathima	12.4	N	83.5	86	24	106	4.6
Babu	8.9	M	77.4	14	13	94	1.7
Anbumani	8.5	N	84.4	14	14	87	1.6
Indrani	10.9	N	97.7	17	30	82	1.7
Sampath	7.8	M	62.5	16	12	96	8.2
Krishnaveni	11.9	N	82.4	11	9	135	1.4
Ravi	8.4	N	102.4	26	33	184	3.6
Davlath Ram	12	N	83.4	24	24	65	3.5
Ravi	10.1	N	91.3	33	25	98	1.7
Indirani	9.8	N	85.4	17	19	98	2.8
Kamala	10	N	90.8	19	22	122	2.2
Duraimani	9	N	80.8	36	25	54	6.6
Saida	10.1	N	99.2	65	75	92	1.6
Valiyammal	8.6	M	75.4	24	28	30	1.9
Goundhan	11.5	N	84.6	18	16	57	4.3
Kalathari	10.2	N	84	33	11	68	1.5
Ravi	8.4	M	75.5	26	33	184	3.6
Rajammal	10.1	N	84.4	24	21	49	2.4
Gangaiammal	5.8	M	72.2	12	11	246	3.6
Chellamal	6.2	N	84	20	20	71	7.1
Gajalakshmi	6.1	M	73.3	17	22	76	1.5
Valliyammal	6.9	N	86.4	35	18	156	2.5
Gangammal	5.8	N	92.1	13	16	179	4.9
Janardhanan	7.2	N	83.6	35	46	26.6	7.9
Velan	9.9	N	85.7	13	11	79	8.5
Ezhumalai	11	M	78.6	125	133	105	1.4
Murugesan	11.3	N	88.6	13	12	36	2.7
Jayaraj	11.3	N	86.4	23	28	123	4.4
Mani	11.9	N	91.6	20	20	100	1.4
Venkatammal	13.2	N	84	30	32	115	2.9
?Arunmiyam	9.2	N	82	16	18	97	3.2
Rani	8.8	M	78	39	86	185	2.7
Rajendran	13.4		86.6	29	14	164	3.2
Ponnamal	14.3	N	82.7	18	16	148	1.7
Mary	13.4	N	84.4	37	16	16	2.2
Muniyammal	8.8	N	83.6	22	18	136	4.1
Jyothi	10.3	N	86.4	32	36	232	3.3
kuppusamy	8.1	N	84	16	12	102	3.4
Mani	9.8	N	82	17	15	97	2.9
Munimmal	9.6	N	85	18	16	112	2.6
Radha	7.2	M	72	26	28	108	3.4
Raniammal	9.2	M	76	14	12	96	1.9
Nagarajan	8.8	N	84	16	13	106	1.8

MASTER CHART

NAME	eGFR estd by COCKROFT GAULT FORMULA	EGFR ESTD BY MDRD FORMULA	eGFR ESTD BY CKD - EPI FORMULA	STAGE OF CKD ACC TO COCKROFT GAULT FORMULA	STAGE OF CKD ACC TO MDRD FORMULA	STAGE OF CKD ACC TO CKD - EPI FORMULA	ACUTE ON CHRONIC KIDNEY DISEASE
Boopalan	23.2962962962963	22.4	20.7	4	4	4	
Kondaiah	46.1309523809524	54.1	52.4	3b	3a	3a	
Perumal	18.6574074074074	21.8	19.4	4	4	4	
Raja	25	20.6	18.9	4	4	4	YES
Vasantha Kumari	15.5087719298246	28.5	27.8	4	4	4	
Ramayi	2.11778262979136	1.6	1.4	5	5	5	YES
Angusamy	4.02298850574713	6.3	5.2	5	5	5	YES
Munusamy	5.31914893617021	6.1	5.4	5	5	5	YES
Parvathy	21.5123456790123	30.5	30.1	4	3b	3b	
Selvi	4.8644578313253	5.2	4.6	5	5	5	YES
Abdul Jaffar	6.98464912280702	7.7	6.9	5	5	5	YES
Ramaih	1.66727582846004	1.6	1.3	5	5	5	YES
Shanthi	6.82206284153005	7.3	6.6	5	5	5	YES
Savitri	6.13275613275613	5.7	5.2	5	5	5	YES
Shenbagavalli	8.21457326892109	6.4	5.8	5	5	5	YES
Parameshwari	7.20338983050847	7.6	6.9	5	5	5	YES
Jagadishwari	5.76608187134503	4.5	4	5	5	5	YES
Selvam	20.8333333333333	29.5	28.3	4	4	4	
Elizabeth	13.1705729166667	15.2	14.0	5	4	5	YES
Subhamma	8.14814814814815	9.2	8.5	5	5	5	YES
Poongothai	7.54079861111111	6.9	6.2	5	5	5	YES
Angaiyan	18.7777777777778	22.6	21.3	4	4	4	
Kuppammal	20.9703947368421	28.2	27.2	4	4	4	
Panjammal	12.6388888888889	14.2	13.0	5	5	5	
Ranganathan	6.00534188034188	5.4	4.7	5	5	5	YES
Vasantha	3.51239669421488	3.3	2.9	5	5	5	YES
Sakunthala	20.5662393162393	19.3	18.0	4	4	4	
Periasamy	31.4542483660131	42.6	40.0	4	3b	3b	
Pandi	40.7407407407407	50.7	49.9	3b	3a	3a	
Ramu	4.50349956255468	4.2	3.5	5	5	5	YES
Chinnaponnu	12.9542042042042	12.9	11.7	5	5	5	
Vazeer Baasha	32.2916666666667	44.7	40.7	4	3b	3b	
Prakash	5.72737686139748	5.9	5.2	5	5	5	
Vasantha	24.8989898989899	24.2	23.6	4	4	4	
Periyasamy	9.91090146750524	11.2	9.6	5	5	5	
Vanitha	26.7480158730159	38.6	35.6	4	3b	3b	
Krishnakumari	18.3823529411765	14.4	13.5	4	5	5	
Murugesan	20.7407407407407	22.8	21.6	4	4	4	
Petchiyammal	20.8188405797101	22.8	22.0	4	4	4	
Gopal	14.161220043573	12.4	11.4	5	5	5	YES
Sunthari	12.3526422764228	11.7	11.0	5	5	5	
Saraswathi	21.5029761904762	25.1	24.1	4	4	4	
Lakshmi	37.3963675213675	43.2	41.8	3b	3b	3b	
Mohammed Hussaun	15.4074074074074	14.2	13.0	5	5	5	YES
Chinnayyan	15.2777777777778	12.8	11.7	5	5	5	
Sambasivam	27.475845410628	30.0	27.7	4	4	4	
Alankaram	20.5507478632479	19.5	18.3	4	4	5	
Egambaram	22.6041666666667	28.0	25.1	4	4	4	YES
Dharman	50	53.3	50.5	3b	3a	3a	
Parthasarathy	9.56709956709957	7.6	6.8	5	5	5	YES

MASTER CHART

NAME	eGFR estd by COCKROFT GAULT FORMULA	EGFR ESTD BY MDRD FORMULA	eGFR ESTD BY CKD - EPI FORMULA	STAGE OF CKD ACC TO COCKROFT GAULT FORMULA	STAGE OF CKD ACC TO MDRD FORMULA	STAGE OF CKD ACC TO CKD - EPI FORMULA	ACUTE ON CHRONIC KIDNEY DISEASE
Dhanapal	4.10526315789474	2.7	2.3	5	5	5	YES
Lakshmi	48.5714285714286	40.8	40.7		3b	3b	
Ellaiyammal	12.2271825396825	11.3	10.4	5	5	5	YES
Ramamoorthy	8.70561282932417	5.9	5.2	5	5	5	YES
Kuppusamy	16.9	12.6	11.5	5	5	5	YES
Dhanalakshmi	25.9473684210526	27.9	26.6	4	4	4	
Poongavanam	19.0828373015873	17.9	16.8	4	4	5	YES
Fathima	11.1382850241546	10.0	9.0	5	5	5	YES
Babu	45.7516339869281	43.9	42.9	3b	3b	3b	YES
Anbumani	31.4765625	34.8	34.4	3b	3b	3b	
Indrani	33.8541666666667	32.1	31.1	3b	3b	3b	
Sampath	8.69749322493225	7.1	6.4	5	5	5	
Krishnaveni	41.8253968253968	40.8	40.7	3b	3b	3b	
Ravi	18.4722222222222	18.2	16.8	4	4	4	
Davlath Ram	15.9920634920635	18.1	15.8	4	4	4	
Ravi	44.4444444444444	43.9	42.9	3b	3b	3b	
Indirani	20.8705357142857	17.6	16.0	4	4	4	
Kamala	27.0454545454545	24.2	23.6	3b	4	4	
Duraimani	11.6792929292929	9.0	8.0	5	5	5	YES
Saida	39.6371527777778	34.8	34.4	3b	3b	3b	
Valiyammal	26.0964912280702	28.2	27.2	4	4	4	
Goundhan	18.1395348837209	14.9	13.8	4	5	5	YES
Kalathari	38.3680555555556	37.0	36.2	3b	3b	3b	
Ravi	19.2013888888889	18.4	17.2	4	4	4	YES
Rajammal	24.3981481481481	21.9	21.2	4	4	4	YES
Gangaiammal	16.724537037037	13.5	12.6	4	5	5	YES
Chellamal	9.54420970266041	6.1	5.3	5	5	5	YES
Gajalakshmi	47.8518518518519	37.6	37.5	3a	3b	3b	
Valliyammal	20.3666666666667	20.7	19.9	4	4	4	YES
Gangammal	13.1519274376417	9.6	9.0	4	5	5	YES
Janardhanan	7.77777777777778	7.4	6.6	5	5	5	YES
Velan	6.06209150326797	6.6	5.7	5	5	5	YES
Ezhumalai	34.7619047619048	53.7	51.6	4	3a	3a	
Murugesan	19.6913580246914	24.7	22.2	4	4	4	
Jayaraj	19.6969696969697	14.7	13.6	4	5	5	YES
Mani	57.1428571428571	54.9	54.2	3a	3a	3a	
Venkatammal	17.9444444444444	17.4	16.4	4	4	4	
?Arunmiyam	22.7864583333333	20.8	19.3	4	4	4	YES
Rani	15.320987654321	18.7	17.5	4	4	4	
Rajendran	19.9131944444444	20.8	19.1	4	4	4	
Ponnamal	33.4027777777778	32.0	30.9	3b	3b	3b	
Mary	16.9570707070707	24.1	23.4	4	4	4	YES
Muniyammal	7.11212737127371	11.3	10.0	5	5	5	YES
Jyothi	20.6060606060606	15.2	14.4	4	4	5	YES
kuppusamy	14.5098039215686	19.4	17.8	5	4	4	
Mani	18.9655172413793	23.1	21.3	4	4	4	
Munimmal	17.9444444444444	19.7	18.7	4	4	4	
Radha	12.5	14.4	13.5	5	5	5	
Raniammal	28.109649122807	28.5	27.8	3B	4	4	
Nagarajan	41.6666666666667	40.4	38.6	3b	3b	3b	

MASTER CHART

NAME	ACUTE PULM. EDEMA	ACIDOSIS	HYPERKALEMIA	ENCEPHALOPATHY	PERICARDITITS	OTHERS	Arterial pH
Boopalan		YES				Hypoglycemia / HYPOKALEMIA	7.24
Kondaiah		YES	YES			A/C exacerbation of COPD	7.24
Perumal		YES		YES		CVA- R MCA INFARCT	7.28
Raja	YES	YES		YES			7.51
Vasantha Kumari		YES					7.35
Ramayi	YES	YES	YES				7.18
Angusamy			YES				
Munusamy		YES		YES			7.39
Parvathy		YES					7.38
Selvi	YES	YES		YES			
Abdul Jaffar	YES	YES					7.42
Ramaih		YES	YES				7.18
Shanthi		YES		YES			7.24
Savitri							
Shenbagavalli	YES	YES	YES				7.20
Parameshwari	YES						7.34
Jagadishwari		YES					7.34
Selvam							
Elizabeth	YES						7.36
Subhamma	YES						
Poongothai	YES	YES		YES			7.33
Angaiyan		YES	YES			PTB POTTS SPINE	7.35
Kuppammal							
Panjammal	YES		YES				
Ranganathan	YES			YES			
Vasantha	YES	YES	YES				7.09
Sakunthala	YES						
Periasamy	YES						
Pandi	YES		YES				
Ramu	YES	YES	YES				7.28
Chinnaponnu			YES				
Vazeer Baasha						Hypokalemia	
Prakash							
Vasantha						Hypokalemia	7.55
Periyasamy							
Vanitha							7.38
Krishnakumari	YES						
Murugesan		YES					7.2
Petchiyammal	YES	YES	YES				7.26
Gopal		YES	YES	YES			7.28
Sunthari		YES		YES			7.25
Saraswathi						Hypokalemia Hyponatremia	7.48
Lakshmi	YES	YES					7.32
Mohammed Hussaun	YES						
Chinnayyan							7.36
Sambasivam							7.49
Alankaram	YES						
Egambaram		YES		YES			7.39
Dharman				YES			
Parthasarathy	YES	YES	YES	YES			7.32

MASTER CHART

NAME	ACUTE PULM. EDEMA	ACIDOSIS	HYPERKALEMIA	ENCEPHALOPATHY	PERICARDITITS	OTHERS	Arterial pH
Dhanapal	YES	YES					7.27
Lakshmi				YES			7.42
Ellaiyammal	YES	YES	YES				7.27
Ramamoorthy	YES	YES					7.3
Kuppusamy		YES					7.32
Dhanalakshmi		YES					7.34
Poongavanam		YES	YES	YES			7.18
Fathima							7.32
Babu		YES					7.34
Anbumani	YES						7.41
Indrani							7.28
Sampath			YES				
Krishnaveni							7.45
Ravi	YES						
Davlath Ram		YES		YES			7.42
Ravi	YES						
Indirani		YES	YES				7.26
Kamala		YES					7.28
Duraimani		YES		YES		Aspiration pneumonitis	7.37
Saida							
Valiyammal	YES						7.39
Goundhan		YES		YES			7.35
Kalathari	YES						
Ravi	YES						7.34
Rajammal	YES	YES	YES				7.38
Gangaiammal							7.38
Chellamal	YES	YES	YES				7.30
Gajalakshmi							7.38
Valliyammal	YES					Uremic gastritis	
Gangammal							
Janardhanan	YES	YES	YES				
Velan	YES	YES	YES	YES			7.38
Ezhumalai		YES					
Murugesan							7.42
Jayaraj	YES						7.37
Mani	YES						
Venkatammal							7.5
?Arunmiyam	YES						
Rani							
Rajendran							
Ponnamal		YES					7.28
Mary							
Muniyammal							7.38
Jyothi							7.48
kuppusamy	YES	YES	YES				7.22
Mani	YES	YES	YES				7.3
Munimmal		YES					7.32
Radha	YES	YES	YES				7.26
Raniammal							
Nagarajan							

MASTER CHART

NAME	pCO2	Bicarbonate	Anion Gap	Serum Sodium	Serum Potassium	Ejection Fraction	LVSD
Boopalan	18	16	22	141	2.8	50%	mild
Kondaiah	52	20	18	140	5.5	60%	-
Perumal	22	16	18	144	4.4	61%	-
Raja	16.7	16.7	20.4	125	4.1	54%	mild
Vasantha Kumari	33.1	17.9	17.9	128	4.9		
Ramayi	16.2	7.7	28.8	128	5.6		
Angusamy				136	5.9		
Munusamy	24.3	16.7	21.8	120	3.1		
Parvathy	30.9	18.7	19				
Selvi				141	3.1		
Abdul Jaffar	24.9	17.8	34.6	134	3.9		
Ramaih	16.2	7.7	25.1	128	5.8		
Shanthi	12.2	10.1	21.6	118	2.8		
Savitri				121	3.7		
Shenbagavalli	29.5	12.6	27.1	130	5.4		
Parameshwari	21.5	11.3	26.2	138	4.4		
Jagadishwari	24.8	13.1	21.3	131	4.8	65%	-
Selvam				133	3.6		
Elizabeth	30.2	17.9	21	139	4.5	65%	-
Subhamma				140	5	48%	mild
Poongothai	25.7	15.1	22	135	4.7		
Angaiyan	15.9	12	22.7	132	5.5		
Kuppammal				133	3.4	63%	0
Panjammal				140	6.4		
Ranganathan				135	5.0	47%	mild
Vasantha	22	7.5	30.7	136	5.7		
Sakunthala				136	5	35%	severe
Periasamy				138	3.6	30%	severe
Pandi				139	5.4		
Ramu	22	14	18	135	6.1		
Chinnaponnu				139	6.2	64%	-
Vazeer Baasha				134	2.2	51%	mild
Prakash				142.6	5.5		
Vasantha	45.3	39.2	32.6	137	1.7		
Periyasamy				142	4.9		
Vanitha	36	22	15	138	4.5	47%	mild
Krishnakumari				159	3.4		
Murugesan	18.3	13.4	22	142	4.4		
Petchiyammal	25.3	13.2	29.4	136	7	50%	mild
Gopal	26	16	16	141	5.7		
Sunthari	28.8	12.2	26	118	4.7		
Saraswathi	30.7	22.6	17.1	113	2.6	70%	-
Lakshmi	14	16	18	142	4.9	50%	mild
Mohammed Hussaun				142	4.1	38%	moderate
Chinnayyan	26	18	21	129	4.1	65%	-
Sambasivam	34.1	26.4	13.2	138	4.9	51%	mild
Alankaram				138	5.1		
Egambaram	29.3	17.3	20.2	122	3.7		
Dharman				136	4.7	65%	-
Parthasarathy	21.5	10.7	24.3	127	5.4	45%	mild

MASTER CHART

NAME	pCO2	Bicarbonate	Anion Gap	Serum Sodium	Serum Potassium	Ejection Fraction	LVSD
Dhanapal	18	8	20	143	4.4	65%	-
Lakshmi	36	22	14	143	4.1	64%	-
Ellaiyammal	22	16	22	130	7.5	47%	mild
Ramamoorthy	24.1	13.4	21.8	138	4.9	60%	-
Kuppusamy	28	18	22	131	4.6	64	-
Dhanalakshmi	25	15.7	28.7	135	4.5	65%	-
Poongavanam	19	14	24	130	5.2		
Fathima	39	20	13.2	130	4.1	66%	-
Babu	35.4	18.8		131	4.2	49%	mild
Anbumani	40	24.6	14.2	142	4.1	41%	moderate
Indrani	42	23	12	138	4	60%	-
Sampath				130	6.2		
Krishnaveni	33.3	22.5	18	132	4.7		
Ravi				141	3		
Davlath Ram	30.0	18	18	137	4.4	55%	mild
Ravi				136	4.3	20%	severe
Indirani	24	14	17	143	5.8		
Kamala	27	12.3	24.5	137	3.9	50%	mild
Duraimani	23.6	13.3	26.5	140	4.4		
Saida				132	3.9		
Valiyammal	19.3	11.4	24.5	145	5		
Goundhan	29.3	17.3	20.8	145	3.6		
Kalathari				125	4.8	60%	-
Ravi	46	19	14	141	3	56%	-
Rajammal	36	26	14.2	143	4.4	60%	-
Gangaiammal	40	18	14.2	141	4.7	60%	-
Chellamal	42	14	16	135	6.6	45%	mild
Gajalakshmi	46	21.2	18.6	138	4.6	46%	mild
Valliyammal				140	3.2		
Gangammal				141	4.7	55%	mild
Janardhanan				145	5.7		
Velan	27	15.4	18.7	139	6.2	63%	
Ezhumalai				141	2.8	48%	mild
Murugesan	34	21.6	10.9	139	4.8		
Jayaraj	24.6	15.7	21.8	130	4.6	50%	mild
Mani				139	3.9	30%	severe
Venkatammal	19.1	20.7	20	123	3.8		
?Arunmiyam				135	4.8		
Rani				133	4.5	70%	-
Rajendran				136	4.1	70%	-
Ponnamal	26	15	16	148	3.4		
Mary				137	2.7		
Muniyammal	36.8	21.0	5.2	140	5.0	60%	-
Jyothi	24	12.9	13.5	138	5.7		
kuppusamy	14	12	18	131	5.8	48%	mild
Mani	22	16	19	132	5.6	45%	moderate
Munimmal	28	16	18	138	4.8	62%	-
Radha	18	12	24	131	5.8	42%	mild
Raniammal							
Nagarajan						42%	moderate

MASTER CHART

NAME	RENAL REPLACEMENT THERAPY	HD / PD / HD AND PD	CENTRE		DIABETES MELLITUS	HTN	CHRONIC GN
Boopalan					YES		
Kondaiah							
Perumal					-	YES	
Raja	YES	HD	SRMC		YES	YES	
Vasantha Kumari					YES		
Ramayi	YES	PD	RGGGH		-	YES	
Angusamy	YES	PD	RGGGH				YES
Munusamy	YES	HD PD	RGGGH		-	YES	
Parvathy					YES	YES	
Selvi	YES	PD	RGGGH		-	YES	
Abdul Jaffar	YES	PD	RGGGH		-	YES	
Ramaih	YES	PD	RGGGH				
Shanthi	YES	PD	RGGGH				
Savitri	YES	PD	RGGGH		-	YES	
Shenbagavalli					YES	YES	
Parameshwari					-		
Jagadishwari					YES	YES	
Selvam					YES		
Elizabeth					YES	YES	
Subhamma					-	YES	
Poongothai	YES	PD	RGGGH		YES	YES	
Angaiyan							YES
Kuppammal					YES	YES	
Panjammal					YES	YES	
Ranganathan	YES	PD, HD	HD-GVMCH PD-RGGGH		YES	YES	
Vasantha	YES	PD	RGGGH		-	YES	
Sakunthala					-	YES	
Periasamy					-	YES	
Pandi					-	YES	
Ramu	YES	PD	RGGGH		YES	YES	
Chinnaponnu					-	YES	YES
Vazeer Baasha					YES	YES	
Prakash					-		
Vasantha					YES	YES	
Periyasamy							
Vanitha					YES		
Krishnakumari					YES	YES	
Murugesan					-	YES	YES
Petchiyammal					YES	YES	
Gopal	YES	PD	RGGGH		-	YES	
Sunthari					YES		
Saraswathi							
Lakshmi					YES		
Mohammed Hussaun	YES	NOT WILLING			YES	YES	
Chinnayyan					YES	YES	
Sambasivam					YES	YES	
Alankaram							YES
Egambaram					YES		
Dharman					YES		
Parthasarathy	YES	HD PD	RGGGH		YES	YES	

MASTER CHART

NAME	RENAL REPLACEMENT THERAPY	HD / PD / HD AND PD	CENTRE		DIABETES MELLITUS	HTN	CHRONIC GN
Dhanapal	YES	PD	RGGGH		YES	YES	
Lakshmi					-	YES	
Ellaiyammal					YES	YES	
Ramamoorthy	YES	PD	RGGGH		YES	YES	
Kuppusamy	YES	PD	RGGGH				
Dhanalakshmi					YES	YES	
Poongavanam					YES		
Fathima							
Babu					YES	YES	
Anbumani					YES	YES	
Indrani					YES	YES	
Sampath					-	YES	
Krishnaveni					YES	YES	
Ravi							YES
Davlath Ram					YES	YES	YES
Ravi					YES	YES	
Indirani					yes	yes	
Kamala					YES	YES	
Duraimani	YES	PD	RGGGH		YES	YES	
Saida					-		YES
Valiyammal					YES		
Goundhan	YES	PD	RGGGH		YES	YES	
Kalathari					YES		
Ravi					-		YES
Rajammal					YES	YES	
Gangaiammal					YES	YES	
Chellamal	YES	PD	RGGGH		YES	YES	
Gajalakshmi					-	YES	YES
Valliyammal							
Gangammal	YES	PD	RGGGH		YES	YES	YES
Janardhanan					-		YES
Velan	YES	PD	RGGGH		YES	YES	
Ezhumalai					-	YES	
Murugesan					-	YES	YES
Jayaraj					YES	YES	
Mani					YES	YES	
Venkatammal					-	YES	
?Arunmiyam	YES	HD	SRMC		YES	YES	
Rani					YES	YES	
Rajendran	YES	PD	RGGGH				YES
Ponnamal					YES	YES	
Mary					-	YES	
Muniyammal						YES	
Jyothi							YES
kuppusamy						YES	YES
Mani					YES	YES	
Munimmal						YES	
Radha					YES	YES	
Raniammal							YES
Nagarajan					YES	YES	

MASTER CHART

NAME	CHRONIC INTERST. NEPHRITIS	MYELOMA	OBS. UROPATHY	ISCH. NEPHROPATHY	OTHERS	UNKNOWN CAUSE
Boopalan						
Kondaiah	YES					
Perumal						
Raja						
Vasantha Kumari						
Ramayi						
Angusamy						
Munusamy						
Parvathy						
Selvi						
Abdul Jaffar						
Ramaih	YES					
Shanthi	YES					
Savitri						
Shenbagavalli						
Parameshwari						YES
Jagadishwari						
Selvam						
Elizabeth						
Subhamma						
Poongothai						
Angaiyan						
Kuppammal						
Panjammal						
Ranganathan						
Vasantha						
Sakunthala						
Periasamy						
Pandi						
Ramu						
Chinnaponnu		YES-MULTIPLE MYELOMA				
Vazeer Baasha						
Prakash					ADPKD	
Vasantha	YES					
Periyasamy						YES
Vanitha						
Krishnakumari						
Murugesan						
Petchiyammal						
Gopal	YES				NSAID NEPHROPATHY	
Sunthari						
Saraswathi	YES					
Lakshmi						
Mohammed Hussaun						
Chinnayyan						
Sambasivam						
Alankaram						
Egambaram						
Dharman						
Parthasarathy						

MASTER CHART

NAME	CHRONIC INTERST. NEPHRITIS	MYELOMA	OBS. UROPATHY	ISCH. NEPHROPATHY	OTHERS	UNKNOWN CAUSE
Dhanapal						
Lakshmi						
Ellaiyammal						
Ramamoorthy						
Kuppusamy						YES
Dhanalakshmi						
Poongavanam						
Fathima						YES
Babu						
Anbumani						
Indrani						
Sampath					ADPKD	
Krishnaveni				YES		
Ravi						
Davlath Ram						
Ravi						
Indirani						
Kamala						
Duraimani						
Saida						?
Valiyammal						
Goundhan						
Kalathari				YES		
Ravi						
Rajammal	YES					
Gangaiammal						
Chellamal						
Gajalakshmi						
Valliyammal						?
Gangammal						
Janardhanan						
Velan						
Ezhumalai						
Murugesan						
Jayaraj				YES		
Mani						
Venkatammal	YES					
?Arunmiyam						
Rani						
Rajendran						
Ponnamal						
Mary						
Muniyammal						
Jyothi						
kuppusamy						
Mani						
Munimmal						
Radha						
Raniammal						
Nagarajan						